

CHANGE YOUR DIET

*A POWERFUL PLAN TO IMPROVE MOOD,
OVERCOME ANXIETY, AND PROTECT MEMORY
FOR A LIFETIME OF OPTIMAL MENTAL HEALTH*

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New York Boston

The advice herein is not intended to replace the services of trained health professionals, or be a substitute for medical advice. You are advised to consult with your health care professional with regard to matters relating to your health, and in particular regarding matters that may require diagnosis or medical attention.

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Balance

Hachette Book Group

1290 Avenue of the Americas

New York, NY 10104

GCP-Balance.com

@GCPBalance

First edition: January 2024

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Library of Congress Cataloging-in-Publication Data

Names: Ede, Georgia, author.

Title: Change your diet, change your mind : a powerful plan to improve mood, overcome anxiety, and protect memory for a lifetime of optimal mental health / Georgia Ede, MD.

Description: First edition. | New York : Balance, 2024. | Includes bibliographical references and index.

Identifiers: LCCN 2023036570 | ISBN 9781538739075 (hardcover) | ISBN 9781538739099 (ebook)

Subjects: LCSH: Mental health—Nutritional aspects. | Nutrition—Psychological aspects. | Mood (Psychology)—Nutritional aspects.

Classification: LCC RC455.4.N8 E34 2024 | DDC 616.85/270654—dc23/eng/20230911

LC record available at <https://lccn.loc.gov/2023036570>

ISBNs: 9781538739075 (hardcover), 9781538739099 (ebook)

E3-20231208-JV-NF-ORI

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To my brilliant partner Suzi Smith for expertly illustrating, co-researching, co-editing, and co-miserating this book with me. Without your ability to help me see the forest for the trees, this manuscript would have been little more than a glorified list of molecules I happen to find fascinating that nobody else cares about.

And to anyone out there who feels like they've tried everything already: please don't give up. If you're willing to try one more thing, hope is on the menu.

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INTRODUCTION

On the night of December 23, 2019, a sudden attack of agitation jolted Karl from a deep sleep.

“I felt like a deer in the headlights.”

Unable to shake the restlessness with his usual relaxation and breathing techniques, he headed out into the night and walked... *for eight miles...* but to no avail. “It was like a startle response that wouldn’t go away.” From that night forward, these unsettling feelings would continue to come and go; they’d leave him in peace for a day or two only to repeatedly revisit him for as long as twenty-four hours, depriving him of sleep and compelling him to walk up to twenty-five miles at a time with little to no relief.

In early February 2020, he sought help from his primary care doctor, who told him his physical exam and blood tests were normal and recommended a melatonin supplement for insomnia. Melatonin did help with sleep, but the agitation simply waited until dawn to overtake him.

The situation had become unmanageable. Karl knew he had to do something, but he was dead set against psychiatric medications. About fifteen years earlier, he’d sought help for mood and attention issues from a private specialty clinic where he underwent a psychiatric evaluation, including sophisticated brain imaging. Thousands of dollars later, he walked out with three psychiatric diagnoses and three prescriptions: Effexor for depression, Klonopin for anxiety, and Adderall for ADHD. After starting low dosages of these medications, he began to feel “super-human, highly focused, and full of energy” and became uncharacteristically arrogant and extroverted. Adding marijuana into the mix to cope with these uncomfortable manic side effects only made the behaviors worse, and he eventually found himself on the brink of divorce.

It was under those dire circumstances that a psychiatrist diagnosed him with bipolar II disorder and urged him to take a mood stabilizer. He instead decided to stop all three medications, joined Marijuana Anonymous, and

found another doctor who was willing to work with his wife to monitor his behavior, agreeing to try a mood stabilizer if any signs of mania re-emerged. From then on, Karl committed himself to a combination of cognitive-behavioral techniques and cycling one hundred miles per week as his “therapy and medication.” This plan was helpful in managing his mood for more than a decade, but it proved no match for the agitation that struck him that December.

Needing a new way forward, Karl turned to the internet and happened upon low-carbohydrate dietary approaches to mental health conditions, so he reached out to me in March 2020 for a nutritional psychiatry evaluation.

His whole life long, Karl had been eating a “standard American diet” full of processed foods, which meant large quantities of refined carbohydrates (sugar, flour, and processed cereals) and refined vegetable oils, so there was plenty of room for improvement. In my mind, there were several dietary strategies worth considering: a paleo diet of meat, seafood, poultry, fruits, vegetables, nuts, and seeds; a whole-foods, low-carbohydrate diet; or even a plant-free “carnivore” diet, which contains virtually no carbohydrate at all. Karl chose the carnivore diet because he hoped it would bring the fastest relief.

At that point, his score on the PHQ-9, a screening test for depression, was 15 on a scale of 0 to 27 (with 27 being the most severe), and his score on the GAD-7, a screening test for anxiety, was 17 on a scale of 0 to 21 (with 21 being most severe).

AN UNORTHODOX REMEDY

After thirty-nine days on his new diet, both his GAD-7 and PHQ-9 scores had fallen to zero. He messaged me: “Just another awesome week without any symptoms of anxiety, agitation, or depression. Nada, zilch, none... yeah!! Overall, I am consistently feeling better than I have for my entire life.”

Psychiatric medications rarely eliminate all symptoms and virtually never produce the empowered joy evident in Karl’s words. After a lifetime of eating a standard American diet, switching to a diet consisting entirely of beef, pork, eggs, and cheese appeared to have completely reversed his mood disorder. Ironically, the only problem he encountered was that despite

eating three to four pounds of fatty animal food per day, he couldn't regain the ten pounds he'd lost during that three-month period of agitation. Therefore, to restore healthy weight, support athletic performance, and add variety, I advised him to relax his diet to include about 100 grams per day of carbohydrate from whole foods—which he did by adding in some plain yogurt and root vegetables like potatoes. After remaining completely well on this plan for over a year, he began broadening his diet to include a greater diversity of whole foods, and for the past year, he has continued to remain well so long as he avoids refined carbohydrates and processed foods and keeps his carbohydrate intake low on days when he doesn't exercise. He is thriving on this simple diet, enjoys eating this way, and remains symptom-free a full three years later.

Whether this remarkable story reflects the unique and irreproducible experience of one man or holds larger lessons that may apply to others, it certainly challenges and inspires us to ask new questions about psychiatry, nutrition science, and the relationship between the two:

- Could some psychiatric illnesses be partly, largely, or even *entirely* dietary in origin?
- What is it about the standard American diet that may be contributing to poor mental health?
- How many people might be able to reduce, avoid, or eliminate the need for psychiatric medications using dietary strategies?

THE ACCIDENTAL NUTRITIONIST

Having practiced psychiatry for more than twenty years, I know all too well the shortcomings of medication-oriented care. Even well into the twenty-first century, the practice of prescribing psychiatric medications continues to be a frustrating trial-and-error process fraught with potential complications. Unpredictable, confusing, and sometimes dangerous drug reactions can occur, especially when more than one drug is started at a time, when drugs overlap during transitions, when drugs are layered on top of each other to manage side effects or address residual symptoms, or when drugs are discontinued too quickly. While skilled, thoughtful use of

medications absolutely improves quality of life for some people and does prevent some hospitalizations, injuries, and suicides, all too often this relief of suffering comes at the expense of side effects such as drowsiness, sexual dysfunction, weight gain, apathy, and high blood sugar.

We can and must do better, and I am convinced that modern nutritional psychiatry is the way forward.

This conviction did not come naturally to me. If you had told me twenty-five years ago that I would be practicing nutritional psychiatry I would have looked at you as if you had three heads. I loved the “hard sciences” of medicine like biochemistry, physiology, and pharmacology and believed that the ability to prescribe medication was the hallmark of a “real doctor.” I viewed the work of nutrition specialists and lifestyle-oriented practitioners with great skepticism.

I would later come to understand that these arrogant attitudes were rooted in sheer ignorance.

Nutrition courses were not required to earn my bachelor’s degree in biology. In four years of medical school, we received only a few hours of nutrition education, and in four years of psychiatric residency training, nutrition wasn’t mentioned once. We were taught that the biological roots of mental illness were due to imbalances in brain chemicals—neurotransmitters such as serotonin and dopamine. I therefore emerged from residency thinking of the brain largely as a bag of neurotransmitters designed to be manipulated with medications, and off I went to the quaint Cape Cod town of Woods Hole to prescribe.

Don’t get me wrong—I was fortunate to have also been taught strong psychotherapy skills from some of the best psychiatrists in the field, so I took thoughtful life histories, explored deeper issues that contribute to emotional distress, and developed meaningful relationships with my patients. During those early years on the Cape, I poured my heart and soul into my work and learned invaluable lessons from the hundreds of people I had the privilege of connecting with who came to me for help. As time went by, however, it became painfully obvious that true healing and full recovery were rare.

Looking around at respected colleagues and mentors, some of whom had been in practice for decades, I noticed the same pattern: Everyone’s practices were filling up with people who weren’t getting better. We met

with patients to provide support, write prescription refills, and try to instill hope, but most of us had quietly come to view mental illnesses as chronic, mysterious, and incurable.

It had never crossed my mind that food might be important to mental health. Like many women, I viewed my own food choices simply as a means of weight control. I ate a low-fat, high-fiber diet largely comprised of skinless chicken breast, fish, vegetables, whole grain cereals, soy milk, hummus, fat-free yogurt, and Diet Coke. I counted calories and exercised religiously. Then, in my early forties, I developed a variety of perplexing new symptoms, including migraines, fatigue, bloating, body aches, and stomach pain. Multiple specialists found nothing wrong, and sophisticated test results were all normal. None of the doctors asked me what I ate, so I left their offices with generic printouts advising me to follow the same low-fat, high-fiber diet I was already eating.

Unwilling to accept these symptoms as my new normal, I started instinctively experimenting with my diet. I began a food and symptom journal and looked for patterns. After about six months of trial-and-error changes, to my complete surprise, I arrived at a highly unorthodox, mostly meat diet, feeling better than I had ever felt in my life. Not only had the pain and fatigue disappeared, but my mood, concentration, and productivity had improved as well. I'd never thought of myself as having much difficulty in these areas, but there was no question that this unconventional way of eating was good for my brain.

As a psychiatrist, I became intensely curious about the relationship between food and brain health and began to wonder whether dietary changes might help some of my patients. As a middle-aged woman, I became concerned that my strange new mostly-meat diet was going to kill me. Since the diet that restored my health was high in animal protein and animal fat, and contained only small amounts of the few plant foods that didn't seem to bother me, my head was full of new questions. Will I get cancer if I don't eat enough vegetables or fiber? Are some fruits and vegetables more important than others, or do I need to eat a wide variety for best results? Which ingredients within red meat make it more dangerous than white meat? How do cholesterol and saturated fat damage the heart—and do they damage the brain as well?

I needed to get to the bottom of these questions, so I started studying

nutrition. In addition to completing a graduate course in human nutrition at the Harvard School of Public Health, I combed the Harvard library database for primary research studies, devouring articles not just about nutrition topics like nutrients, digestion, and metabolism, but also about botany, anthropology, toxicology, animal husbandry, and agriculture. What I discovered was that *nearly everything I thought I'd known about nutrition was wrong*.

RETHINKING BRAIN FOOD

I was genuinely shocked to learn that there is absolutely no science (or logic) behind recommendations to eat plant-based diets, balanced diets, high-fiber diets, low-cholesterol diets, or diets containing whole grains, low-fat dairy products, or rainbows of fruits and vegetables. At best, these ideas represent well-intentioned guesses based on deeply flawed, unscientific food questionnaires; at worst, they are intentional distortions of the facts designed to protect professional reputations or serve political and commercial agendas, not to protect and serve public health.

The truth about nutrition is this: Meat is not dangerous, vegan diets are not healthier, and antioxidants are not the answer. So, where can we look for answers?

The good news is that hiding underneath that mountain of biased, confusing guesswork are clear, elegant, compelling scientific principles about nutrition that make intuitive sense, work in clinical practice, and stand the test of time. Do we know everything we wish we knew? No. Do we know more than enough for you to substantially improve your brain health starting today? Absolutely.

Most of us have been feeding our brains improperly our entire lives, therefore we have no idea how much better we can feel and how much more we can expect of ourselves if we eat right.

Most books about nutrition and mental health ask you to pin your hopes on plant superfoods (which do not work) and supplements (which often profit the author), often without showing you how to improve the overall nutritional quality of your diet from the ground up in ways that will minimize your need for supplements. These books often also recommend Mediterranean diets or plant-based diets for optimal brain health without

explaining the very real risks of these dietary strategies. It saddens me to see so many people working so hard to make good food choices, not realizing those choices are based on bad information—information that can damage the brain over time and *increase* risk for serious problems with mood and memory. By focusing on the right whole foods, customizing your carbohydrate intake to suit your metabolic needs, and eliminating common food sensitivity culprits, you can greatly improve not only your mental health, but your whole health.

I believe that many of the emotional and cognitive issues we have come to expect as normal, genetic, or permanent can be prevented, eased, or even reversed with good nutrition. If you don't want to take medication, don't respond to medication, can't tolerate medication, or can't access medication, there are innovative dietary strategies you probably haven't tried yet that can help medication work better, counteract certain medication side effects (such as weight gain), or in some cases reduce or even eliminate the need for psychiatric medication.

My goal in writing this book was to take the confusion out of nutrition and replace it with science, simplicity, and common sense; to teach you how to think for yourself about food so you can make your own informed choices and find what works best for you and your family.

A NEW WAY FORWARD

This book is divided into four parts.

In part 1, I'll show you how sloppy, unscientific research methods have led to flip-flopping headlines, illogical guidelines, and public confusion about what we're supposed to eat. The problem is that most brain food researchers study nutrition from the *outside in*, by questioning people about their eating habits and then trying to guess how their food choices might be affecting their mental health. This flawed approach is why some of us dutifully top our morning oatmeal with blueberries, choose plant-based patties over hamburgers, or wash handfuls of supplements down with kale smoothies. We're told that these habits will protect our brains, but not only are these strategies very unlikely to help, they can even work against us. In this book, we will look at nutrition from the *inside out* by discovering what the brain needs to function at its best and then using that list of ingredients

to redefine what a brain-healthy diet should look like.

In part 2, we'll explore the dietary roots of our global mental health crisis. You'll see exactly how our modern ultraprocessed diet contributes to brain inflammation, hormonal imbalances, neurotransmitter imbalances, emotional instability, depression, and dementia—and how focusing on the right whole foods and customizing your carbohydrate intake can restore internal harmony and reveal your best self. For those of you who need more relief, there is an entire chapter dedicated to the promise of ketogenic diets for psychiatric disorders.

In part 3, I take you on a guided tour of the fascinating world of food. We'll weigh the risks and benefits of different foods groups, learn how they affect the brain, and sort out which ones are essential and which ones are optional so you can make informed choices about what to eat. I'll introduce you to some of the devilishly clever natural chemicals lurking within grains, legumes, nightshades, and certain other plant foods that can work against optimal brain nutrition and function, but I will also help you identify kinder, gentler plant foods so you can find the mix that works best for you.

In part 4, I boil down all of the information laid out in previous sections into three dietary strategies—all of which can be customized to your food preferences, health circumstances, and personal goals. Since changing how we eat is hard, you'll find meal plans and recipes for each one, along with plenty of tips and tools to support your success. I am nutritionally pro-choice and want everyone to have a seat at the table, so regardless of your dietary preferences, you will find the information you need to optimize your diet for better mental health.

My hope is that this book will ignite your curiosity about food and the brain, empower you and your family to live happier, healthier lives, and bring you peace of mind.

PART 1

RETHINKING BRAIN FOOD

CHAPTER 1

What Causes Mental Health Problems?

Every solution to every problem is simple. It's the distance between the two where the mystery lies.

—Derek Landy, *Skulduggery Pleasant*

We are in the midst of a global mental health crisis.

Nearly one billion people are living with a mental health disorder,¹ including one in five of the world's children and adolescents.² Every year, 700,000 people take their own lives, and suicide is now the second leading cause of death among people in their teens and twenties. Depression and anxiety alone cost the global economy nearly three billion dollars a day.³ And these numbers don't include the countless people with milder mental health concerns like brain fog, irritability, and joylessness. Psychiatric problems of all kinds are becoming so commonplace that we are beginning to think of poor mental health as normal and inevitable.

Between 2007 and 2018, while I was serving as a psychiatrist at Harvard University and then at Smith College, my seasoned colleagues and I observed a most disturbing trend: It was becoming increasingly common for first-year students to arrive on campus already taking one, two, or even three psychiatric medications. Requests for specialized support for learning and emotional disabilities were rising so fast that it was difficult to accommodate everyone's needs. More and more students were showing up at campus mental health clinics in crisis, requiring emergency psychiatric hospitalizations, leaves of absence, or academic withdrawals. The sense among clinicians on the front lines is that the mental health of our young people is increasingly brittle, and research supports our observations.

According to a 2018 American College Health Association report, more than 40 percent of students "felt so depressed they had difficulty

functioning,” and more than 60 percent had experienced “overwhelming anxiety.”⁴ A 2018 study conducted by the American Association for Suicidology observed a nearly tenfold increase in non-suicidal self-injury among first-year college students over only a seven-year period.⁵ In the UK, declarations of existing mental health problems among university students have risen by a staggering 450 percent in just the past decade.⁶

Crumbling mental health isn’t just a problem among young people on college campuses. Ohio State University Professor Hui Zheng conducted a study across nine generations, from the Greatest Generation (born between 1900 and 1924) to Generation Y (born between 1981 and 1999), and observed that both the mental and physical health of all generations born since the 1950s has been declining across all sex and racial groups.⁷

If you are a fellow mental health professional, you don’t need statistics to tell you how challenging things have become. Everywhere I’ve worked—clinics, hospitals, universities—I’ve encountered the same issues: Practitioners overwhelmed by large, complex caseloads, and patients frustrated with wait times that are too long for appointments that are too short. Administrators try to ease the burden by hiring more staff, offering group appointments, and training peer counselors, but there never seems to be enough time or resources to meet the growing needs of the people we are trying to serve. It’s like trying to fight a wildfire one teaspoon of water at a time. Meanwhile, everyone is working so hard that there’s no time to stop and ask: Why is our mental health deteriorating? Is there anything we can do about it, or do we simply accept it as inevitable?

If we are to have any hope of reversing this tragic trend, we need a better understanding of the root causes of psychiatric disorders.

IN SEARCH OF UNDERSTANDING

The brain is our most mysterious organ. Sequestered deep within the skull and possessing no nerve endings, we can’t see it, touch it, or feel it working, so questions about what causes mental illness and unwellness have baffled us for millennia. Some ancient civilizations believed that those suffering from mental illness were possessed by demons or being punished by God for their sins, calling for spiritual treatments such as exorcism and prayer. In the Middle Ages, psychiatric symptoms were blamed on a buildup of vile

bodily fluids that needed to be relieved with leeches or laxatives.⁸

By the mid-1900s, these beliefs had given way to theories about the root causes of mental illness that continue to dominate our thinking today: stress, childhood trauma, chemical imbalances... and, of course, your mother.

These theories have their merits but are ultimately unsatisfying.

The Stress Factor

In the early 1800s, it was thought that people with mental illness had inherited incurable weaknesses that left them unable to adapt to the mounting stresses of a rapidly industrializing society. As prominent British psychiatrist Dr. Henry Maudsley wrote in 1867, “an increase of insanity is a penalty which an increase of our present civilization necessarily pays.”⁹ As a result, most nineteenth-century psychiatrists served largely as stewards of asylums, where people could be sheltered from the daily pressures of modern living. Without effective treatments, residents in their care were not expected to improve and lived out their lives on the grounds of psychiatric hospitals.

Could the pressures of today’s lifestyle—social media, injustices related to race and gender identity, growing economic inequality, and gun violence, to name a few—help explain the decline in mental health we are currently experiencing? Perhaps, but are the stresses of our time really more challenging than those of Maudsley’s era? One could argue that the world has always been a stressful place. And just as industrialization, globalization, and information technology pose new challenges that make some aspects of our lives more stressful, they also bring new conveniences that make other aspects of our lives such as transportation and communication less stressful. Stress certainly can contribute to poor mental health, but stressful obstacles are part and parcel of daily life. The question is: Why do some of us embrace new challenges while others struggle to face them?

The Mind-Brain Divide

Most nineteenth-century psychiatrists appeared to have little interest in brain biology, so it would be neurologists who would first explore this new

scientific frontier. Locked away in their chambers of bone, the brains of the living defied direct examination, so neurologists of the 1800s focused their microscopes instead on the brains of the departed. By studying specimens from individuals with speech impediments and other obvious neurological conditions, early neurologists learned enough about brain anatomy in only a couple of short decades to begin creating a map of its functions. However, when they inspected the brains of former asylum residents, they couldn't identify any structural abnormalities—these brains looked completely normal.¹⁰

Psychiatrists would therefore enter the twentieth century trying to understand the mind rather than the brain, relying on their powers of observation and imagination to diagnose and treat mental illnesses. It was during this period that Austrian neurologist Dr. Sigmund Freud developed his influential theory that psychiatric suffering arose from repressed fantasies and traumatic early childhood experiences buried deep in the unconscious that could be unearthed through psychoanalysis, founding a branch of psychiatry that continues to thrive to this day. I value modern talk therapy and have been incorporating it into my clinical work for more than twenty years, but I have yet to see psychotherapy alone put any case of serious mental illness into remission.

The Psychiatric Medication Revolution

The biological branch of psychiatry didn't emerge in earnest until the 1930s and 1940s, with the accidental discovery of a number of experimental treatments for schizophrenia and other serious mental illnesses. These strange and horrific new interventions, which included insulin coma therapy, the lobotomy, and a primitive, crude form of ECT (electroconvulsive therapy), did help some people, but injured (and sometimes killed) many more, causing all of these methods to eventually fall out of use. These desperate tactics are just a few of the many inhumane skeletons in psychiatry's closet, so when psychiatric medications burst onto the scene in the mid-twentieth century,¹¹ they were welcomed with open arms.

These early drugs included lithium—a long-forgotten mineral with mood-stabilizing properties¹²—and chlorpromazine, the first antipsychotic

medication. Originally developed in France in 1952 to calm patients before surgery, chlorpromazine (marketed under the brand name Thorazine) proved useful in reducing agitation, delusional thinking, and hallucinations in some individuals with schizophrenia.

Psychiatrists accustomed to relying on physical restraints and other undignified methods of keeping people safe and calm experienced chlorpromazine as nothing short of revolutionary. As Dr. Robert Cancro, then chair of psychiatry at the New York University School of Medicine, reflected in 2000: “It is difficult to communicate to younger colleagues the miracle that 150 to 300 mg of chlorpromazine a day appeared to be to the house officers [psychiatry residents] of 1956.... Finally, we were like other doctors in that we had a treatment that actually worked. It was truly an intoxicating time.”¹³

Researchers believed that chlorpromazine worked by blocking the activity of dopamine—a neurotransmitter that brain cells use to communicate with each other. The novel idea that emotional and behavioral problems could be caused by chemical imbalances in dopamine, serotonin, and other neurotransmitters captured the imaginations of clinicians and the general public alike. This exciting new neurotransmitter theory of mental illness pulled psychiatry out of the dark ages and into the modern medical age. Over the ensuing decade, a firehose of pharmaceuticals would be aimed at everything from major mental illnesses to the minor stresses of everyday life. These innovative chemicals included clozapine (Clozaril) for psychosis, imipramine (Tofranil) for depression, methylphenidate (Ritalin) for hyperactivity, diazepam (Valium) for anxiety, and meprobamate (Miltown) for nervousness. Although rarely prescribed today, meprobamate was a trailblazing tranquilizer that first normalized the practice of taking pills to ease minor psychological discomfort. As Dr. Jerome Groopman wrote in *The New Yorker*, “Approved in 1955, meprobamate (marketed as Miltown and Equanil) was hailed as a ‘peace pill’ and an ‘emotional aspirin.’ Within a year, it was the best-selling drug in America, and by the close of the fifties one in every three prescriptions written in the United States was for meprobamate.”¹⁴

Those pioneering drugs of the 1950s and 1960s are all still around. In fact, although many new drugs have been developed since then, none of them work in truly new ways; they are all just safer or reimaged versions

of the originals.¹⁵ Even cutting-edge treatments like transcranial magnetic stimulation (TMS) and psychedelic-assisted therapies, such as ketamine and psilocybin treatments, target neurotransmitter imbalances in the brain.

Strengths and Weaknesses of Standard Psychiatric Care

Since the 1950s, the neurotransmitter theory of mental illness has dominated the landscape of biologically minded psychiatrists, whereas the stress and trauma theories have continued to prevail among psychosocially minded psychiatrists, but all psychiatrists are trained to take all of these theories into consideration. We are taught that it is your unique stew of biological, psychological, and social ingredients that produces your thoughts, emotions, and behaviors, and it is this *biopsychosocial* model of the origins of mental illness that we have in mind when we meet with you for the first time to conduct a one-hour standard psychiatric evaluation. In addition to asking about your symptoms, we also ask about your family history, medical history, relationships, worldview, and your work and home environment, to create a three-dimensional impression of your life that puts your symptoms into context.

To make a formal psychiatric diagnosis (which insurance companies require), we turn to a 1,000-plus-page reference book called the *DSM (Diagnostic and Statistical Manual of Mental Disorders)* to see if your symptoms match any of the hundreds of diagnoses it contains. Even if you happen to fit neatly into any of its official diagnostic boxes, the *DSM* offers no guidance about how we should treat your symptoms, let alone any biological clarity about what might be causing them.

In the absence of clear treatment guidelines, we use the information gathered during your interview to formulate a biopsychosocial theory of your case—essentially, an educated guess about what might be causing your symptoms. We use this to develop your personalized treatment plan, which often includes medication (to address chemical imbalances) and some form of counseling, such as psychotherapy (to process stressful life experiences) or cognitive-behavioral therapy (to change negative thought and behavior patterns).

A real strength of the biopsychosocial model is that it values your human story—a story that psychiatrists believe plays a major role in your

emotional and physical well-being, and that medical professionals in other fields may not have time to explore. Most psychiatrists I know, myself included, truly enjoy this aspect of the work. We love paying quality attention to all the little details and nuances of your history, piecing them together, and sharing impressions that we hope you will find helpful. Most people who come to us for help also enjoy the process and find therapeutic value in being seen, heard, and understood on a level that goes beyond symptom lists and diagnostic tests. These precious intangibles of the biopsychosocial model are what set psychiatry apart from other branches of medicine and make it such a rich and rewarding profession. However, a serious shortcoming of this approach is that our current diagnostic framework lacks the biological specificity we need to be confident in the medical elements of our assessments and treatments. The main difference between psychiatrists and other mental health professionals is that psychiatrists are medical doctors, and therefore we are uniquely qualified to assess and treat the “bio” elements of your biopsychosocial story—the biology behind your symptoms—yet this is the piece we understand the least.

We are taught that some people are born with differences in genes and neurotransmitters that make them more susceptible to depression, psychosis, or severe anxiety—particularly when under extreme stress or after suffering a traumatic life experience. Yet, even as we enter the second quarter of the twenty-first century, we still have no telltale genetic tests to offer you and no reliable way to measure your brain’s neurotransmitter activity. The brain has a separate circulatory system, so we can’t evaluate its biochemistry by drawing blood from your arm and running simple laboratory tests. These obstacles to understanding the inner workings of your brain leave us little choice but to resort to guesswork when making medication recommendations. In comparison to other fields of medicine, the practice of psychiatry still feels like more of an art than a science; we can’t tell you what is causing your symptoms, so we can’t tell you which medication is most likely to help.

Another challenge we face is that psychiatric medications don’t work as well as we’d like. The best studies available find that approximately 50 percent of people with depression benefit from standard antidepressants—which sounds good, until you learn that approximately 40 percent of people

improve with placebo alone.¹⁶ Furthermore, the degree of improvement is minuscule (on average, an increase of a mere two points on a fifty-two-point depression symptom scale), and more than half of clinical trials find no benefit at all.¹⁷

Medications prescribed for bipolar disorder and schizophrenia perform better, but still leave too many without meaningful relief. One-quarter of people with serious mental illness benefit from antipsychotic medications, which is about twice as many as improve with placebo alone.¹⁸ Approximately one-third of people with bipolar disorder respond to mood stabilizers,¹⁹ but nearly half of those who initially experience relief from medication-supported interventions continue to experience recurrent mood episodes despite continuing treatment.²⁰ Why are so many people “treatment-resistant”? Are they failing treatment, or is treatment failing them?

The fact that medications let so many people down tells us that neurotransmitter imbalances represent only one small piece of the biological puzzle. We must be missing something, because seventy-plus years of sophisticated pharmaceuticals engineered specifically to target neurotransmitter imbalances have clearly failed to stem the tide of our growing global mental health crisis.

There are times when psychiatric medication can be life-changing and even lifesaving. If you are in crisis, the right medication could help you hold on to your job, stabilize a fragile relationship, stay in school, keep you out of the hospital, or even prevent you from taking your own life. Unfortunately, the price you pay for these benefits may include side effects that reduce your quality of life, such as drowsiness, sexual dysfunction, or dulled emotions; and side effects that reduce your length of life such as obesity, cardiovascular disease, and type 2 diabetes.

It’s not that there is no truth whatsoever to the neurotransmitter theory of mental illness; neurotransmitters play important roles in our mood, memory, and concentration circuits. The question is: What causes neurotransmitters to become unbalanced in the first place?

To improve the safety and effectiveness of our treatments, we need to better understand what is happening inside the brains of people with mental illness. We now have sophisticated modern imaging techniques that use magnets or radiation to peer inside the brain and observe its chemistry in

action, but these are complicated, expensive, and invasive tests not available to most people, and we are only beginning to understand how to interpret their findings. Fortunately, while we await further advances in neuroscience research to help us get better at zooming in on the brain's inner workings, there is much we can learn by zooming out and reminding ourselves that *the brain is part of the body*.

As Goes the Body, So Goes the Brain

Just as our mental health has been spiraling downward in recent decades, so, too, has our physical health.

In the United States, cases of heart disease nearly doubled between 1990 and 2019,²¹ and the percentage of Americans with obesity has nearly tripled since the 1960s.²² Globally, the percentage of adults with type 2 diabetes doubled between 1980 and 2016, and body weight has been steadily rising; between 1975 and 2015, obesity rates worldwide more than doubled among women and more than tripled among men.²³ People with obesity, type 2 diabetes, and cardiovascular disease are also far more likely to have psychiatric disorders like depression, bipolar disorder, and schizophrenia, and this is no coincidence.

While all of these physical and mental health conditions may seem unrelated to each other, they commonly occur together and share many of the same underlying abnormalities, the most important being *inflammation, oxidative stress, and insulin resistance*.²⁴

Inflammation and oxidative stress are part of your immune system's first responder network, so it is normal and healthy to have a certain degree of both, but *excessive* inflammation and oxidative stress can be very damaging to every cell in the body—and brain cells are no exception.

Insulin resistance (which is often called “prediabetes”) is a common metabolic disorder in which insulin doesn’t work as well as it should. If you have insulin resistance, your body will need to produce more than the usual amount of insulin to try to keep your blood sugar (and brain sugar) levels stable and in a healthy range, so your insulin levels will tend to run too high. Over time, high insulin levels can make it more difficult for your brain to turn glucose (blood sugar) into energy.

It just so happens that our industrially ultraprocessed diet is a powerful

promoter of inflammation, oxidative stress, and insulin resistance—all of which are just as dangerous for the brain as they are for the rest of the body. In the long search for biological root causes of mental illness—a search that has been focused almost exclusively on neurotransmitters for nearly seventy-five years—*inflammation, oxidative stress, and insulin resistance* have emerged as an unholy trinity of destructive forces that help to explain why those neurotransmitter imbalances occur.

We readily accept that diet plays a major role in the health of the rest of the body—why should the brain be any different? The foods we eat provide the construction materials we need to build healthy, resilient brain cells and the fuel we need to energize them. If we don't eat the right foods, none of our cells will develop or function properly, and any number of things can and will go wrong—including many things no medication can address.

Medications can and do change brain chemistry, and they have their place, but I'm convinced that *the most powerful way to change brain chemistry is through food, because that's where brain chemicals come from in the first place*. Neurotransmitters are made from food, the brain cells that pass them back and forth to communicate with each other are made from food, and even the salty soup that surrounds them is made from food. Optimal mental health requires that your *whole brain* be made of the right stuff, so if you have a mental (or physical) health problem of any kind, the first place to look isn't your medicine cabinet, it's your pantry. This advice holds true whether you view mental health conditions as primarily biologically driven or psychosocially driven, because, as we'll see in the coming chapters, the way we eat has a profound impact on brain development, neurotransmitters, stress hormones, inflammation, antioxidant capacity, brain energy production, brain aging, and brain healing.

There is only so much you can do to reduce your exposure to stress, and nothing you can do to change the genes you were born with or the childhood you experienced, but you can change your diet—and changing your diet can change your mind.

CHAPTER 2

The New Science of Hope

People are fed by the food industry, which pays no attention to health, and are healed by the health industry, which pays no attention to food.

—Wendell Berry, *Sex, Economy, Freedom, & Community*

Our diet has undergone radical changes in the past century. Born in 1910 and raised in a rural New England farm town, my grandmother ate two soft-boiled eggs and buttered toast every morning for breakfast, ground her own hamburger with a medieval-looking device she clamped to the kitchen counter, and kept an old coffee can full of bacon fat by the stove for cooking. By the time she passed away in 1993, all three of these foods were falling out of favor with the American public and had been officially condemned as dangerously unhealthy. The first U.S. Dietary Guidelines, released in 1980, warned that saturated fat and cholesterol caused obesity and heart attacks, so they advised Americans to “moderate your use of eggs,” “limit your intake of butter,” and “trim excess fat off meats.”¹

Food manufacturers sought to capitalize on these new food rules by flooding the market with fat-free sweets and cholesterol-free fats like corn oil, canola oil, and margarine.² By blaming modern health epidemics such as obesity, type 2 diabetes, and heart disease on saturated fat and cholesterol, the U.S. Dietary Guidelines drove us away from nutritious whole foods like meat and eggs and right into the arms of the ultraprocessed food industry. Like a perfect storm, the powerful forces of food industrialization, growing anti-meat sentiment, and fat and cholesterol phobia collided and have been feeding on each other for the past fifty years, dramatically transforming our nutritional way of life. Since most other nations pattern their food guidelines after the U.S. guidelines, this shift

away from animal fats and toward refined carbohydrates and vegetable oils meant that the whole world was about to take part in a grand nutrition science experiment—with devastating consequences.

Characterized by an abundance of calorie-dense, nutrient-poor foods and beverages,³ the so-called *standard American diet* (I'll take the liberty of referring to this as the “SAD diet” going forward), isn’t just an American problem anymore—this modern atrocity has been exported to all four corners of the Earth, endangering the physical and mental health of people everywhere.

Unfortunately, we don’t have much concrete information about the mental health of people prior to the modernization of our diet, but what little we do have suggests that our mental health was more robust in the past than it is today.

Industrial globalization has made it difficult to locate people in this century who eat entirely off the land, but in the middle of the last century, there were still pockets of dietary sanity to be found. In a 2003 paper titled “Nutrition and Schizophrenia,” University of Sheffield psychiatrist Dr. Malcolm Peet highlighted interesting studies from Taiwan, Tonga, Trinidad, Papua New Guinea, Malawi, and Australia’s Gold Coast, all of which suggested that schizophrenia was far less common in people who fed themselves by hunting, fishing, and subsistence farming.⁴ As Dr. Peet wrote: “It is remarkable that studies of truly indigenous populations are virtually unanimous in reporting very low rates of schizophrenia.”⁵ For example, signs of schizophrenia were exceedingly rare among non-Westernized Pacific Islanders in the 1950s. Of 60,500 inhabitants examined, researchers identified only two individuals with psychotic behavior (0.003 percent), whereas the prevalence of psychosis among Europeans of the same time period was sixty-seven times higher (0.2 percent).⁶

Of course, food isn’t the only difference between modern Western ways of life and the lifestyles of these Indigenous groups, and observations of this nature don’t represent hard evidence of a connection between modern diets and our mental health crisis; unfortunately, that level of evidence doesn’t exist. It is simply food for thought: Perhaps serious mental illnesses don’t need to be as common as they have become.

NUTRITIONAL PSYCHIATRY AND THE MEDITERRANEAN DIET: A BETTER WAY FORWARD?

The relatively new specialty of *nutritional psychiatry* was established on the belief that the deterioration in the quality of our diet is largely to blame for the deterioration in our mental health.

For the prevention and treatment of depression and other mental health conditions, most thought leaders within this budding field recommend changing from the SAD diet to the *Mediterranean diet*. Although vaguely and inconsistently defined, the Mediterranean diet has recently been described as being:

- high in whole grains, vegetables, fruit, nuts, legumes, and olive oil
- moderate in seafood, poultry, eggs, low-fat dairy, and red wine
- low in sweets, red meat, and processed meats⁷

The story of how the Mediterranean dietary pattern and its familiar “whole grains good, animal fats bad” philosophy became implanted in our collective psyche is told masterfully by investigative journalist Nina Teicholz in *The Big Fat Surprise*.⁸

Part wishful thinking, part wild guess, the Mediterranean dietary pattern essentially began as a romantic theory about what we should eat, inspired by cherry-picked aspects of cherry-picked Mediterranean traditions, and propped up by unscientific studies conducted in the 1950s and 1960s by Dr. Ancel Keys, a University of Minnesota researcher who believed that saturated fat caused heart disease. (We will see what makes studies like his unscientific in [chapter 3](#).)

The creators of the Mediterranean diet didn’t start with a thoughtful examination of the nutritional risks and benefits of individual foods, use that information to design a dietary pattern, and then test that pattern in human clinical trials to see if it improved health. Instead, they observed that people living in countries along the north shore of the Mediterranean Sea generally seemed to be healthier than Americans, assumed that some of the differences in the way they ate must be responsible for their superior health, and then designed a dietary pattern that they thought represented the

healthiest aspects of those culinary traditions. Among the important revelations in Teicholz's book is that Professor Walter Willett (a prominent nutrition researcher who was chairman of the Harvard School of Public Health at the time) prematurely declared the Mediterranean diet to be a healthy eating pattern in 1993—seven years before the diet would first be tested in human clinical trials.⁹

The Mediterranean diet has since been extensively tested in dozens of human clinical trials for physical health conditions such as obesity, cardiovascular disease, and type 2 diabetes, and has consistently outperformed the SAD diet, earning the trust of the medical community and nutrition policymakers alike. As for mental health conditions, although studies testing the Mediterranean diet's potential to prevent or treat memory and cognitive health issues have produced mixed results,¹⁰ three clinical studies have now demonstrated that switching from a poor-quality SAD diet to the Mediterranean diet can improve symptoms of clinical depression when added to standard psychiatric treatment (medication and/or psychotherapy).¹¹ The science is clear: The Mediterranean diet is healthier than the SAD diet, so if you currently eat a SAD diet, switching to the Mediterranean diet would be a solid step in the right direction.

What makes the Mediterranean diet healthier than the SAD diet? Is it the nuts? The olive oil? The red wine? We really don't know. Those who advocate for the Mediterranean diet *speculate* that it is superior to the SAD diet because it is lower in saturated fat, trans fats, and added sugars; richer in essential nutrients; higher in fiber; and chock full of colorful fruits and vegetables brimming with *phytonutrients*—naturally occurring plant chemicals believed to have unique anti-inflammatory and antioxidant properties.¹² However, there are so many differences between these two dietary patterns that there is no easy way to determine which aspects of the Mediterranean diet are responsible for its health benefits.

Almost any change you make to the modern atrocity that is the SAD diet is bound to make it healthier. In other words, just because emerging evidence supports the idea that the Mediterranean diet is *better* for the brain than the SAD diet doesn't necessarily mean that it is the *best* diet for the brain—and there are good reasons to suspect that it is not. A few examples:

- The grains and legumes that form the foundation of the Mediterranean diet are nutrient-poor themselves, and even contain *antinutrients* that interfere with our ability to access certain essential minerals.
- The Mediterranean diet frowns on some sources of refined carbohydrate such as sweets, while celebrating others, such as bread and pasta.
- The Mediterranean diet encourages the consumption of alcohol.

But the Mediterranean diet's biggest blind spot is that it pays far too little attention to metabolic health. In other words, it contains too much carbohydrate for people with insulin resistance to safely process, resulting in higher insulin levels that can damage brain metabolism over time. The word “metabolism” refers to the complicated collection of chemical reactions our cells use to turn food into energy. Since the brain is an energy hog, if its metabolic machinery can't generate enough power to meet its needs, it can and will malfunction.

Metabolic Health Is the Missing Link

After decades of standing still, the field of psychiatry is taking a quantum leap forward. A revolutionary new way to think about mental health is changing the way scientists study mental illness, transforming the way psychiatric professionals approach clinical care, and empowering individuals and families to improve their mood, concentration, and memory—often reducing or even eliminating the need for psychiatric medication. The breakthrough realization of our time is that robust metabolic health is essential to robust mental health.

Within only the past five years or so, an exciting new subspecialty of psychiatry has emerged called *metabolic psychiatry*. This term was coined by Stanford University psychiatrist Dr. Shebani Sethi, who defines it as “a new subspecialty focused on targeting and treating metabolic dysfunction to improve mental health outcomes.”¹³

Investigators in this field are discovering that what many psychiatric conditions have in common is that the brain has trouble burning glucose for energy. One of the most important obstacles to brain energy flow is insulin

resistance, a serious metabolic disorder that is reaching epidemic proportions in many places around the world. Diets too high in refined carbohydrates like sugar, flour, fruit juice, and cereal products promote the persistently high insulin levels that lead to insulin resistance. Therefore, as counterintuitive as it sounds, the more sugar your diet contains, the harder it becomes for your brain to use it.

High blood glucose and insulin levels are a deadly one-two punch for the brain. Repeatedly flooding the brain with too much glucose triggers unrelenting waves of inflammation and oxidative stress, damaging your brain's delicate architecture and overwhelming its *mitochondria*—the tiny engines inside your cells that work tirelessly to turn glucose into energy. Repeatedly bombarding the brain with too much insulin can lead to insulin resistance, which makes it increasingly difficult for insulin to enter the brain where it is needed to help turn that glucose into energy. The high-glucose, low-insulin brain struggles to generate the power it needs for peak performance, resulting in a slowly mounting brain energy crisis.

The empowering news is that you can control your glucose and insulin levels yourself—and rather quickly, too—simply by changing what you eat.

The Ketogenic Diet Is Powerful Metabolic Medicine

Reimagining mental health disorders as metabolic disorders has opened the door to exciting new treatment approaches, the most powerful of which is the *ketogenic diet*.

Ketogenic diets are very-low-carbohydrate, moderate-protein, high-fat diets that stimulate your body's ability to burn fat—and to turn some of that fat into *ketones*, which your brain can burn for energy. For brains that have lost some of their ability to use glucose properly, ketones are a godsend, because they help bridge the energy gap created by brain glucose processing problems.

Some people think of the ketogenic diet as a weight loss diet—and yes, it can help you burn excess body fat. Some people think of the ketogenic diet as a diet for type 2 diabetes—and yes, it is a very effective way to treat type 2 diabetes because it reliably lowers blood glucose levels, reduces or eliminates the need for diabetes medications, and can even put type 2 diabetes into sustained remission.¹⁴ But did you know that the original

intended purpose of the ketogenic diet was to stabilize brain chemistry?

In 1921, long before anticonvulsant medications were available, the ketogenic diet was invented to treat children with epilepsy. More than a dozen quality clinical trials have since demonstrated that ketogenic diets are safe and effective in both children and adults with epilepsy, cutting seizure activity by more than half in more than 50 percent of cases, and completely eliminating seizures in 10 percent or more.¹⁵ The ketogenic diet has shown promise in many other neurological conditions as well, including multiple sclerosis, Parkinson's disease, and migraine headaches.¹⁶ This strong evidence base in neurology is good news for psychiatry, because, as I see it, the line between neurology and psychiatry is *imaginary*. The brain isn't divided into neurology cells and psychiatry cells—it is one organ. It stands to reason that if a particular treatment benefits *neurological* brain disorders, it should also benefit *psychiatric* brain disorders. Emerging evidence looks very promising, and scientific interest in this area is suddenly exploding.

Psychiatric conditions *are* neurological conditions—it's just that their symptoms were historically considered to be more psychological than biological. A powerful case in point is bipolar disorder, a serious mood disorder that includes periods of mania (unusually high energy) that are typically followed by periods of deep depression. Bipolar disorder and epilepsy share many common underlying features—in fact, many of the medications we prescribe to treat bipolar disorder are anti-seizure medications. If a ketogenic diet can stabilize seizures in people with epilepsy, it stands to reason that it may also stabilize mood swings in people with bipolar disorder, and this is the line of reasoning that led me to begin incorporating ketogenic diets into my clinical work more than ten years ago. So, is the ketogenic diet the best diet for the brain?

We could attempt to answer this question by testing ketogenic diets against Mediterranean diets, vegetarian diets, SAD diets, and the countless other dietary patterns one can imagine, but this would be a daunting proposition, so instead, let's take a step back and ask a different question: What does a brain-healthy diet need to accomplish?

WHAT IS A BRAIN-HEALTHY DIET?

I propose that for any dietary pattern to be considered brain-healthy, it must

fulfill all three of the following criteria:

1. It must NOURISH the brain by including adequate amounts of all essential nutrients.
2. It must PROTECT the brain by excluding damaging ingredients.
3. It must ENERGIZE the brain in ways that support healthy metabolism for a lifetime by keeping blood sugar and insulin levels in a healthy range.

These are the principles I used to create the dietary strategies I share with you in this book, and these very same principles apply to the rest of the body as well. All of our cells require the same nutritional care, which is fortunate, as eating a different diet for every organ we possess would be inconvenient, to say the least.

The Mediterranean diet nourishes the brain better than the SAD diet, but it could be even more nutritious. It also protects the brain better than the SAD diet because it discourages ultraprocessed foods and limits certain types of refined carbohydrate, but it could be even safer.

Ketogenic diets reliably lower glucose and insulin levels, so they are very effective at addressing insulin resistance, and they excel at energizing the brain because they generate ketones that the brain can use as a supplemental fuel source, but their ability to nourish and protect the brain can vary tremendously depending on food choices.

The Quiet Diet Approach

The limitations of these dietary patterns led me to create three new dietary patterns for you to explore: Quiet Paleo, Quiet Keto, and Quiet Carnivore.

I call these diets “quiet” because their food lists have been uniquely modified to target root causes of mental health conditions by quieting inflammation, oxidative stress, and high insulin levels. These plans are also lower in natural irritants and toxins and easier to digest than the standard paleo, keto, and carnivore diets you may already be familiar with, so they are much less likely to bother you if you have food sensitivities or poorly understood health conditions such as chronic fatigue, fibromyalgia, and irritable bowel syndrome. Recognizing that not everybody wants or needs to adopt a ketogenic diet to improve their mental health symptoms, I’ll show you how to figure out where you stand on the insulin resistance spectrum, help you customize your carbohydrate intake to your metabolic

needs, and present you with a variety of other brain-healthy changes to choose from.

Quiet Paleo: Like standard paleo, this plan allows meat, seafood, poultry, eggs, fruits, and vegetables and excludes grains, legumes, dairy, refined carbohydrates, alcohol, vegetable oils, and ultraprocessed foods. (These excluded foods jeopardize brain health by interfering with nutrient access, disrupting energy flow, and promoting inflammation and oxidative stress.) However, a key difference is that Quiet Paleo limits plant foods to a special list of what I call “kinder, gentler” fruits and vegetables that are not only lower in sugar, but also lower in the chemicals that plants use to protect themselves from predators.

Quiet Keto: Like all ketogenic diets, this plan is very low in carbohydrate, moderate in protein, and high in fat, but Quiet Keto is based on the Quiet Paleo food list (minus the higher-carbohydrate fruits and vegetables), so it combines nutritional quality with metabolic quality, giving you the best of both worlds.

Quiet Carnivore: Like most carnivore diets, this plan is plant-free (and therefore free of plant toxins), but Quiet Carnivore also discourages common animal food sensitivity culprits like eggs, dairy, and processed meats. Quiet Carnivore can be particularly helpful if you have unrecognized food sensitivities, gut damage, autoimmune conditions, and other stubborn or mysterious syndromes that haven’t responded to other dietary interventions.

After the initial discovery phase, if you find that your mental health has improved on one of these plans, you can then try expanding your food list to find the least restrictive and most enjoyable diet you can tolerate.

Change Your Diet, Change Your Mind

It breaks my heart to see people struggling with mental health problems who have tried a dozen or more medications or years of psychotherapy but have never tried changing their diet in any way. Or worse yet, to see people faithfully following what they’ve been told is a brain-healthy diet of whole grains, legumes, nonfat dairy, blueberries, dark chocolate, and red wine yet continuing to feel depressed, confused, anxious, or unstable—believing they are already doing all they can, when there is SO much more they can

do. Whether you are dealing with a serious mental illness like schizophrenia, or an everyday mental health concern like irritability, I want you to know there is hope.

Changing your mind *with* diet requires changing your mind *about* diet. And the first step on that journey is to clear your mind of any preconceived notions you may have about food so we can start fresh. Let's take a closer look at where mainstream beliefs about nutrition come from so you can decide for yourself if they are deserving of public trust.

CHAPTER 3

Why Most Nutrition Guidelines Are Wrong

Published and true are not synonyms.

—Brian A. Nosek, Jeffrey R. Spies, and Matt Motyl, “Scientific Utopia”

The good ship *Salisbury* had a grave problem on her hands. In the spring of 1747, she had left the port of Plymouth, England, with a crew 300 men strong.¹ After just eight short weeks at sea, at least 30 of her sailors had contracted scurvy, a disease that killed an estimated two million mariners between the sixteenth and nineteenth centuries.² In what may have been the world’s first controlled trial in clinical nutrition on record, naval surgeon Dr. James Lind took it upon himself to conduct a simple experiment. As he tells it:

I took twelve patients in the scury, on board the *Salisbury* at sea. Their cases were as similar as I could have them. They all in general had putrid gums, the spots and lassitude, with weakness of their knees... and had one diet common to all... water-gruel sweetened with sugar in the morning; fresh mutton-broth often times for dinner; at other times puddings, boiled biscuit with sugar, and for supper, barley and raisins, rice and currants, sago and wine, or the like.³

He divided the twelve men into six pairs, and administered the following treatments to see which, if any, might be helpful:

Pair #1: One quart of cider per day

Pair #2: Twenty-five drops of dilute sulfuric acid three times per day
Pair #3: Two spoonfuls of vinegar three times per day
Pair #4: One half-pint of seawater per day
Pair #5: Two oranges and one lemon per day
Pair #6: A medicinal paste of garlic, mustard seed, radish root, balsam sap, and gum myrrh

Per Dr. Lind:

The consequence was, that the most sudden and visible good effects were perceived from the use of oranges and lemons; one of those who had taken them, being at the end of six days fit for duty.... The other was the best recovered of any in his condition; and being now deemed pretty well, was appointed nurse to the rest of the sick.⁴

Lind's results provided evidence that fresh citrus fruits could cure scurvy. Almost 400 years later, his protocol still stands as a fine example of the scientific method. Defining the scientific method can be surprisingly difficult, but its bedrock principles can be summarized in this definition of science itself, taken from the Oxford English Dictionary: "the systematic study of the structure and behavior of the physical and natural world through *observation and experiment* [emphasis mine]."⁵

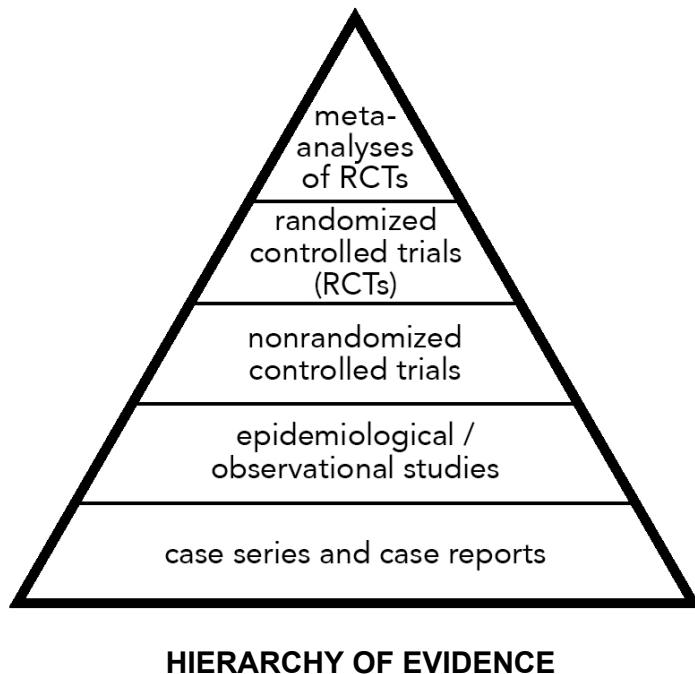
We would have to wait nearly two centuries for Hungarian scientist Dr. Albert Szent-Györgyi to identify the curative chemical stowed within those oranges and lemons as vitamin C, which earned him a Nobel Prize in 1937. This juicy revelation was just one of many vitamin discoveries made during the so-called "Vitamin Era" of the 1930s and 1940s, all made possible by the Chemical Revolution—the development of laboratory techniques that allowed researchers to isolate and study vital food molecules for the first time in human history.⁶

When President Eisenhower suffered a heart attack in 1955, public fear of cardiovascular disease shifted the focus of diet research away from micronutrients (vitamins and minerals) to macronutrients—fat and cholesterol, to be exact—ushering in the era of politicized nutrition in

which we still find ourselves today. In an attempt to understand how complex dietary patterns cause or prevent things like heart attacks, nutrition research largely lost its way in the second half of the twentieth century, veering away from solid scientific methods grounded in experimentation in favor of a wholly unscientific method grounded in guesswork called *nutrition epidemiology*. Most mainstream views about food and health (such as the belief that plant foods are healthier for us than animal foods) spring from nutrition epidemiology studies, so it's important to understand the serious shortcomings of this type of research and how it compares to other nutrition research methods.

MODERN NUTRITION RESEARCH METHODS

Studying the relationship between diet and disease is no easy task, and all nutrition research methods have their shortcomings, but some methods are far more reliable than others. When you see a nutrition headline, knowing something about the kind of study that was used to generate that headline can help you quickly decide if the headline is worth your attention. Scientists sometimes disagree about which types of evidence are most reliable, but the pyramid below represents a common ranking order.



Let's begin at the bottom of the pyramid and work our way to the top.

A nutrition **case report** describes how a particular dietary intervention (such as a ketogenic diet) affected the health of a single patient, while a **case series** describes how a dietary intervention affected the health of multiple patients who all have a similar health condition—for example, how a ketogenic diet affected five people who all have early Alzheimer's disease. Well-documented case reports often contribute valuable information that inspires additional research,⁷ but since they are not formal scientific experiments, these sit at the bottom of most evidence pyramids.

In **nutrition epidemiology** (aka “**observational**”) studies, researchers gather information about large numbers of people and analyze that information to look for patterns that may explain health trends in communities. An example within the world of nutrition would be conducting a survey of thousands of people about their egg yolk eating habits and their heart health history, and then sifting through their answers to see if there might be a connection between the number of egg yolks they reported eating and whether or not they develop heart disease. The lion’s share of nutrition studies that make headlines are epidemiological studies, perhaps in part because they are inexpensive and relatively easy to conduct.

A **nonrandomized controlled trial** is a scientific experiment in which volunteers with similar health conditions are divided into two groups—an *experimental group* (which changes their diet) and a *control group* (for comparison). For example, to test the effects of cholesterol-rich egg yolks on blood cholesterol levels, you might feed everyone in the experimental group two egg yolks per day and feed everyone in the control group something similar that you wouldn’t expect to have any impact on cholesterol levels—such as two egg whites per day, which are cholesterol-free. At the end of the experiment, you compare the cholesterol levels of the two groups to see if the egg yolks seemed to make a substantial difference. These trials are called nonrandomized because the decision about which volunteers join which group isn’t made randomly; instead, the researchers or the volunteers themselves decide who will belong to each group, which could influence the results. (Dr. Lind’s scurvy experiment would therefore be considered a nonrandomized controlled trial, as he himself decided which sailors received which treatment.)

Just below the pyramid’s pinnacle is the **randomized controlled trial** or

RCT. Widely regarded as the gold standard of scientific research methods, the RCT is considered the best way to explore whether there is a cause-and-effect relationship between two items of interest or *variables* such as egg yolks and cholesterol levels. Instead of the researchers or the volunteers themselves choosing who will be on Team Yolk and who will be on Team White, volunteers are randomly assigned to each group, usually by a computer, reducing the chance that human bias will influence the results. The best-designed RCTs are *double-blinded*, meaning that neither the volunteers nor the researchers know who is in the experimental group and who is in the control group.

Proudly perched atop the pyramid pinnacle is the **meta-analysis**, which pools the results of multiple RCTs and analyzes them as a group to look for trends.

A well-designed RCT makes it easier to conclude that a particular dietary intervention is directly responsible for the results that the researchers observe because it attempts to minimize other factors that could muddy the waters. For example: if one of my patients tries a ketogenic diet for early Alzheimer's disease and she happens to score better on memory tests six weeks later, a scientific journal may agree to publish my findings in the form of a case report—especially if this is the first case of its kind. However, because the circumstances weren't controlled, I can't claim that the ketogenic diet was responsible for my patient's improvement. Maybe she made other changes during that same six-week period that she didn't mention or that I chose not to include, such as eliminating junk food or taking a multivitamin. Maybe she was expecting the ketogenic diet to help because a family member had responded well to it. (People who believe in a treatment are more likely to experience benefits—this is called the placebo effect.) Maybe I had ten patients with Alzheimer's who tried the diet, and she was the only one who improved—but I didn't report the other nine cases because I wanted to share the most hopeful news.

RCTs seek to minimize variables like these that can muddy the waters. For example, when researchers in New Zealand conducted an RCT comparing the ketogenic diet to a low-fat diet in people with Alzheimer's disease, they randomly divided twenty-six volunteers into two groups using randomizing software, gave them all the same dietary instructions, and told them all to take the same multivitamin supplement. They told the volunteers

that both diets were potentially healthy, and volunteers weren't allowed to tell researchers which diet they were following during the study.⁸ These design elements made it more likely that the improvements the researchers documented in quality of life and ability to function were due to the ketogenic diet and not due to other variables such as vitamin supplementation or volunteer expectations. In other words, it's more likely that the ketogenic diet itself *caused* those improvements to occur.

Nutrition RCTs: All That Glitters Is Not Gold

Despite their coveted position within the pyramid, even randomized controlled trials have their limitations, particularly when used to study food.⁹ RCTs are used all the time by pharmaceutical companies to test whether a particular drug is safe and effective compared to a placebo, but they're not used very often to study human nutrition because diet RCTs are so expensive and difficult to design.

The control problem: It is impossible to study only one dietary variable at a time because you can't make one change to a diet without also changing something else. For example, if Team Yolk eats two egg yolks a day and Team White eats two egg whites a day, cholesterol content won't be the only difference between their diets—Team White will also be eating fewer calories, fewer nutrients, and less fat. These additional variables or *confounders* make it challenging to design appropriate control conditions for nutrition RCTs and to interpret their results.

The blinding problem: In drug RCTs, researchers can disguise the medicines (and placebos) they want to test in unmarked capsules, but it is very difficult to blind volunteers in nutrition RCTs, especially if you're studying foods rather than individual nutrients or ingredients. Vitamins and food chemicals can be hidden in a capsule or stirred into a beverage, but how do you disguise a whole food like a banana? Diet study participants can usually see, smell, and taste the food being tested, which could influence how people think and behave in the study.

The time and place problem: It is difficult to control and document everything volunteers eat unless you admit them to a metabolic research ward where all their meals can be prepared in a scientific kitchen and staff can record everyone's actual food intake. This is inconvenient and stressful

for volunteers and expensive for researchers, making rigorous long-term diet experiments like this highly impractical. For all of these reasons, it is common for researchers to conduct nutrition RCTs in animals or in test tubes instead of in human beings.

It's much easier to control animals and laboratory samples than it is to control human beings, which helps address time, place, control, and blinding problems, but when you move out of the real world and into carefully controlled laboratory settings, you introduce entirely new problems.

Animal RCTs Must Be Interpreted with Caution

The most obvious problem with conducting nutrition RCTs in animals is that animals aren't humans, and different species require different diets.

In 1913, Nikolai Anichkov, a young Russian pathologist interested in studying the relationship between dietary cholesterol and heart disease, wondered if dietary cholesterol could raise blood cholesterol, build up in the arteries, and cause heart disease. To test this hypothesis, he fed one group of rabbits sunflower oil (the control group), fed another group of rabbits sunflower oil plus purified cholesterol (the experimental group), then measured their blood cholesterol levels and examined their arteries. From his writing:

The blood of such animals exhibits an enormous increase in cholesterin [sic] content, which in some cases amounts to several times the normal quantity. It may therefore be regarded as certain that in these experimental animals large quantities of the ingested cholesterin are absorbed, and that the accumulations of this substance in the tissues can only be interpreted as deposits of lipoids circulating in large quantities in the humors of the body.¹⁰

Put simply: Rabbit cholesterol levels skyrocketed—often to ten times their normal levels or higher—and fatty deposits appeared in their blood vessels within a matter of weeks. Anichkov's experiments are now

considered classic, because they appeared to show, for the first time, a clear connection between dietary cholesterol, rising blood cholesterol levels, and cardiovascular disease. But did they?

Rabbits are herbivores. They did not evolve to consume animal foods, which are the only foods that contain cholesterol. When other investigators tried to reproduce Anichkov's findings in omnivores such as rats and dogs, cholesterol levels barely budged and arteries remained clear, presumably because omnivores are equipped to safely process incoming cholesterol molecules. Careful scrutiny of rabbit arterial deposits found they bore little resemblance to human atherosclerotic plaques, and there's no mention of any rabbit having suffered a heart attack. Rabbits continuing with cholesterol treatment did perish, but not from heart disease; they ultimately developed fatty cirrhosis of the liver, hemolytic anemia, and wasted away: "The cholesterol-fed rabbits exhibit anorexia, lassitude, progressive and severe weight loss, and fur thinning... the animals eventually die in a cachectic state."¹¹

These century-old experiments do not demonstrate that cholesterol endangers the human heart, they demonstrate that *cholesterol is poisonous to rabbits*. This inconvenient truth renders them a very poor choice of subject for cholesterol research, to say the least. Nevertheless, many cholesterol scientists would come to revere Anichkov's work and revive his techniques, putting rabbits back on the methodological menu, where they remain to this day.

Problems with animal research go beyond species differences.¹² While some types of animal research can generate information relevant to human health (indeed, we have laboratory animals to thank for much of what we understand about basic brain cell biology), we must interpret animal studies with caution, particularly when studying the relationship between nutrition and human disease. Laboratory animals are caged in extremely stressful artificial environments and fed ultraprocessed, species-inappropriate diets. Inbreeding is commonplace, and many animals have been genetically or pharmaceutically manipulated to more easily develop diseases of interest like diabetes or cancer.

In Vitro Nutrition RCTs Are Rarely Relevant

In vitro (Latin for “in glass”) nutrition studies examine the effects of isolated food chemicals on living cells or tissues in test tubes, Petri dishes, and the like. These types of studies are frequently used to test extracts from plant foods believed to have superfood properties, such as broccoli, blueberries, and beets. Unfortunately, these experiments tell us very little about nutrition and human health because we do not place food chemicals directly onto all of our cells in plastic containers—we swallow those chemicals in the form of food. Not only do living cells behave very differently when they are removed from the human body and grown in a laboratory, but many food chemicals don’t survive digestion, aren’t absorbed into the bloodstream, or are rapidly eliminated by our immune system. Just because a substance might kill cancer cells in a Petri dish doesn’t mean it will shrink tumors in the living human body.

NUTRITION EPIDEMIOLOGY: A BRIDGE TOO FAR

Every research method has its soft spots, but, if thoughtfully designed and responsibly interpreted, all of them are capable of generating information that can improve our understanding of the relationship between food and human health. Even *uncontrolled* trials can be valuable.¹³ For example, if Lind had fed citrus fruits to all 30 scurvy-stricken men—without any control group for comparison—and none of them had improved, that would certainly have been worth reporting. Indeed, every methodology within the pyramid has the potential to produce meaningful data—even most types of epidemiological studies—the notable exception being epidemiological studies of *human nutrition*. In this section, I aim to convince you that any and all claims about food and human health that have been generated by nutrition epidemiology studies can and should be completely ignored.

Epidemiological studies typically sit near the middle of most evidence hierarchy pyramids, but this placement overlooks the fact that not all epidemiological studies are created equal. Epidemiology can be useful when studying infectious diseases or even vitamin deficiencies, but I would argue that epidemiological studies of the relationship between dietary patterns and human health don’t belong in the pyramid at all because they are wholly unscientific.

The field of epidemiology (the study of epidemics) was born in the mid-

1800s, with many crediting its origins to British physician John Snow. During a deadly cholera outbreak in the Soho district of London, Dr. Snow suspected that polluted city water might be to blame. To explore this hypothesis, he interviewed townspeople about their water usage habits and meticulously mapped out where infections had occurred. He noticed a striking pattern: Most infected households were clustered around a city water pump located on Broad Street.



An 1854 map of the Soho neighborhood of London with public water pumps indicated by encircled P's and cases of cholera infections indicated by black dashes. Notice that most cholera cases are clustered near the Broad Street water pump.

John Snow, On the Mode of Communication of Cholera (London: Churchill, 1855), map 1.

This *association* between proximity to the Broad Street pump and cholera infections was so strong that it convinced skeptical city officials to remove the handle from the Broad Street pump. When locals could no longer draw water from that pump, the epidemic came swiftly to an end, confirming Dr. Snow's hypothesis.

Epidemiology has since proved useful for understanding other diseases also caused by *single, quantifiable toxins* such as cigarette smoke and COVID-19. The epidemiological method of studying disease is considered *observational* because it relies on systematic analysis and pattern recognition instead of on clinical experiments—after all, it would be

unethical to intentionally expose healthy people to potentially lethal bacteria, viruses, or tobacco.

More than a century after Dr. Snow's landmark cholera study, nutrition researcher Professor Walter Willett, whom we met in the previous chapter, began applying the observational methods of epidemiology to the study of diet and chronic disease. While he was not the first to use this approach (Dr. Ancel Keys, whom we also met in the previous chapter, used epidemiology to study the relationship between saturated fat and heart disease), Willett is considered by many to be the founding father of *nutrition epidemiology*. He wrote an authoritative textbook on the subject, has co-authored more than 1,700 research papers on nutrition and public health, and his work continues to wield tremendous influence around the world.

If epidemiology is good enough to help us understand and fight rapid-onset, deadly diseases caused by infections and toxins, then why shouldn't it also be good enough to help us understand and fight slow-onset, chronic diseases that may be driven by diet, such as type 2 diabetes, cardiovascular disease, and Alzheimer's dementia?

First of all, there's only one thing that can cause cholera, and that is cholera toxin. In sharp contrast to this clear, direct relationship, as Willett himself wrote, chronic diseases "almost always have multiple causes... not only diet, but also genetic, occupational, psychosocial, and infectious factors; level of physical activity; behavioral characteristics... and other influences."¹⁴

Secondly, Dr. Snow focused squarely on two concrete variables: where people got their water and whether they got sick—two questions easy for any household to answer with a high degree of certainty. Diet, on the other hand, as acknowledged by Willett himself, "represents an unusually complex set of exposures that are strongly intercorrelated" and patterns of consumption vary significantly over time.¹⁵ As we will see shortly, it is impossible to obtain concrete data about people's diets, and without data, science can't happen.

This fatal flaw hasn't deterred nutrition epidemiology from taking flight. Since the 1940s, nutrition epidemiologists have been generating hypotheses about foods and human diseases, asking people about their eating habits, and looking for patterns in their answers to see if they can find associations between specific foods and specific diseases to support their hypotheses.

For example, if a research group has hypothesized that the cholesterol in egg yolks causes heart disease, and finds that people who report eating more egg yolks are also more likely to develop heart disease, they write a paper about the association they have observed and publish it in a scientific journal. Perhaps not surprisingly, most readers believe that academic papers like these contain scientific evidence that egg yolks endanger heart health, but this is not the case.

Remember, the scientific method requires two steps: generating a hypothesis and then conducting an experiment. Dr. Snow used the data he gathered about water sources and cholera infections to hypothesize that water from the Broad Street pump was making people sick, but he didn't stop there. Next, he convinced local officials to do an experiment—remove the Broad Street pump handle to see if his theory held water, so to speak. Since cholera infection rates dropped sharply, the experiment supported Snow's hypothesis.

Nutrition epidemiologists do not change the diets of people, animals, or flasks of cells to see what happens. In fact, they conduct no experiments of any kind. *The absence of experimentation renders nutrition epidemiology a wholly unscientific discipline.* Good science is self-correcting and moves us closer to the truth. If you never put your theory to the test, you will never know how right or wrong it may be; if it is wrong, avoiding experimentation allows you to continue believing in your theory without ever having to re-evaluate it. Indeed, many popular theories put forth by nutrition epidemiology fly in the face of human biology, yet are allowed to stand.

Sadly, most of our nutrition beliefs have been built on these bio-illogical pillars of sand. As an example, let's take a closer look at a seemingly impressive study that is largely responsible for the belief that dutifully topping your oatmeal with blueberries every morning will help ward off dementia.

This Is Your Brain on Berries

A Harvard nutrition epidemiology research group was interested in whether the antioxidants in colorful berries could help protect aging women against memory loss. Over fourteen years, they studied the eating habits of more

than 16,000 middle-aged women. Then, over the course of six additional years, they periodically tested them for signs of memory problems. Applying sophisticated statistics to their observations, they calculated that women who reported eating two or more servings of strawberries and blueberries per week had a slower rate of memory decline than those who reported eating fewer berries. Voila! They found an *association* between berries and memory protection.

Because they are Harvard researchers who studied many thousands of subjects over twenty years, and their findings have the potential to improve the lives of countless women around the world, their work was published in the prestigious journal *Annals of Neurology*: “Dietary Intake of Berries and Flavonoids in Relation to Cognitive Decline”¹⁶ and enjoyed widespread media attention, generating high-profile headlines like these:

“Eating Blueberries and Strawberries Staves Off Memory Decline, Study Suggests” —CBS News¹⁷

“Brain Food: Berries Can Slow Cognitive Decline” —*Time* magazine¹⁸

“Berries Keep Your Brain Sharp” —Harvard Gazette¹⁹

Unfortunately, because this study is a nutrition epidemiology study, it can’t tell us anything about how berries affect brain health. Why not?

Memories Are Not Measurements

Science demands data. Data, by definition, must be *objective and quantifiable*. In our berry study example, researchers did not record what people actually ate over fourteen years (this would be virtually impossible). Instead, they administered a “semi-quantitative food frequency questionnaire” (FFQ) to inquire about the dietary habits of participants. Below is the berry question from this study’s FFQ—how accurately can you answer this question?

Please fill in your *average* use *during the past year* of each specified food.

(Please try to average your seasonal use of foods over the entire

year. For example, if a food such as cantaloupe is eaten 4 times a week during the approximate 3 months that it is in season, then the average use would be one per week.)

frozen, or canned (½ cup)								
Blueberries, fresh, frozen, or canned (½ cup)								
Peaches, apricots, or plums (1 fresh or ½ cup canned)								

Frank E. Speizer et al., “Nurses’ Health Study Questionnaire (1984 Long),” Nurses Health Study, <https://nurseshealthstudy.org/participants/questionnaires>.

This survey asks you to recall how often you have eaten specific serving sizes of various fruits over the course of the *entire previous year*. If you only eat a particular fruit when it is in season, you are even asked to do the math to convert your seasonal fruit intake into an annual average. You are not given the option of responding “I don’t know,” “I can’t remember,” or “You can’t be serious”—you’re required to enter a specific quantity, even if you’re not sure.

It is wholly unrealistic to expect anyone to recall with any certainty what they’ve eaten over the past twelve months when most people probably can’t recall what they’ve eaten over the past twelve days. Scientists have long recognized that human memory is fallible. In 1984, Bernard et al. reviewed questionnaire-based studies across a variety of disciplines and concluded that “on average, about half of what informants report is probably incorrect in some way.”²⁰

Human memory is also subject to conscious and unconscious distortion. Some people may believe they eat healthier than they actually do, while others may know they eat poorly and feel shame around their food choices; these feelings may influence their answers. When 240 adults were asked how they would handle diet questions if they were to participate in a nutrition study, 29 percent acknowledged they would not answer questions honestly, and 46 percent said they did not think they’d be able to answer questions accurately.²¹ As a result, instead of neutral, objective measurements, food frequency questionnaires produce distorted, subjective estimates.

To make matters worse, the researchers themselves do not appear interested in accuracy. Notice the vague and arbitrary serving sizes in their berry question—“one slice” of this, “one small glass” of that. These are *meaningless, unscientific quantities*. Can you imagine a laboratory chemist trying to follow an experimental protocol that calls for “one small glass” of hydrochloric acid? As Harvard chemistry professor E. B. Wilson Jr. stated in his textbook *An Introduction to Scientific Research*, “A measurement whose accuracy is completely unknown has no use whatever.”²²

Indeed, the very name of the questionnaire used in this study—“semi-quantitative FFQ”—tells us that inaccuracy is baked right into the study’s methodology, as the term “semi-quantitative” means “constituting or involving less than quantitative precision.”²³

Food Frequency Questionnaires Are Too Infrequent

Using a single questionnaire to capture an entire year’s worth of food choices is bound to result in grave errors; using a single questionnaire to capture several years’ worth of food choices borders on absurdity. When researchers conduct a nutrition epidemiology study that spans many years or decades, they do not administer FFQs as often as once a year. In our berry study example, questionnaires were administered only five times over the course of fifteen years; researchers then simply averaged the five sets of answers together to arrive at total berry intake. During the ensuing six-year period when memory tests were conducted, researchers didn’t inquire about food intake at all. Even if the FFQ were a reliable means of gathering data, the idea that berry intake between 1980 and 1994 would be solely responsible for any memory problems that arose between 1995 and 2001, and that eating habits between 1995 and 2001 would have no impact on memory is challenging to accept. Imagine a physician trying to apply research findings like this in clinical practice to help patients concerned about cognitive decline:

Patient: Doctor, I seem to be having more trouble remembering things lately.

Doctor: Well, Barbara, research has shown that memory problems can be due to berry deficiency. How many half-cup servings of

strawberries and blueberries did you eat per week ten years ago?

Modern Diets Are Too Complex for Epidemiology

In the 1990s when this berry study was initiated, a typical U.S. supermarket stocked approximately 7,000 products,²⁴ yet the food frequency questionnaire used in this study considered only 130 food items—less than 2 percent of what was available to shoppers at the time. As Stanford epidemiology professor John Ioannidis noted in his 2018 article “The Challenge of Reforming Nutritional Epidemiologic Research,” “Individuals consume thousands of chemicals in millions of possible daily combinations. For instance, there are more than 250,000 different foods and even more potentially edible items, with 300,000 edible plants alone.”²⁵

The fruit question reveals a failure to attempt to capture this degree of complexity, as only fifteen fruits are represented. The number of blackberries, cherries, kiwis, papayas, figs, mangos, dates, pineapples, honeydew melons, plantains, raspberries, and cranberries these women ate was apparently considered unimportant.

Proponents of nutrition epidemiology willing to acknowledge its flaws rightly point out that RCTs have their limitations too, and that using RCTs to explore the long-term effects of complex dietary choices on human health is not feasible. They argue that nutrition epidemiology is uniquely capable of studying large populations over many decades and taking multiple dietary variables into consideration. Professor Willett and colleagues wrote in 2015:

Because of its low cost and low participant burden, self-administered computer-processed FFQs are the only option in most large cohort studies to assess usual dietary intakes.... These features make possible repeated assessments over time, which is important to capture longer term variation in diets.... Nutritional epidemiology is far from being a perfect science, but with a thorough understanding of the discipline, valuable insights on diet and health outcomes can be obtained from free-living populations.²⁶

In other words, we are asked to forgive the inaccuracies in their measurements and appreciate the information made possible by nutrition epidemiology studies, flawed though it may be, because it represents the best we can do. Yet data collection issues are only the tip of the iceberg.

Association Is Not Necessarily Causation

Even the most thoughtfully designed epidemiology study is only capable of documenting hypothetical associations between a potential culprit and a particular disease, not of establishing cause-and-effect relationships between the two. Without putting your theory to the test in the real world, all you have is a hunch. Dr. Snow's cholera theory was just a theory until the water pump handle was removed. If people who report eating more berries also appear to suffer less cognitive decline than people who don't, this doesn't necessarily mean that berries have anything to do with it—the relationship between the two could be pure coincidence.

If people who report eating more pretzels are also more likely to suffer from alcoholism, that doesn't necessarily mean pretzels cause alcoholism—it could simply mean that people who drink too much spend more time in bars where free pretzels are served. Yet it would be completely acceptable in the field of nutrition epidemiology to publicize that untested association using language that implies a causal relationship between the two, in a manner such as this: "Eating Pretzels Increases Risk of Alcoholism."



Suzanne Smith

I like to use this imaginary example because the idea that pretzels might cause alcoholism is so preposterous that it's easy to see the flawed leap in logic and dismiss the headline as invalid. By contrast, if an association is found between two things we've been conditioned to believe are connected —such as red meat and cancer—we are far more likely to take the headline at face value. In other words, believing is seeing.

The associations observed by epidemiologists outside of the nutrition realm certainly can and have been used to help demonstrate a cause-and-effect relationship between two variables. Sir Austin Bradford Hill, a professor of medical statistics and epidemiology (and the first scientist to randomize a clinical trial), offered nine “viewpoints” to help epidemiologists decide when an association may suggest causation, which are: strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment, and analysis. First introduced in his classic 1965 paper, “Environment and Disease: Association or Causation?,”²⁷ these so-called *Bradford Hill criteria* are now widely regarded as instructive considerations in the field, including by Professor Walter Willett.²⁸

A discussion of all nine criteria is beyond the scope of this book, so we'll focus on the first and most important of Professor Hill's criteria,

which was that associations should be *strong*.

Epidemiologists often express the strength of their associations in terms of *relative risk*. Using smoking and lung cancer as an example, nonsmokers are the unexposed control group and smokers are the exposed group. Relative risk tells you how much more often lung cancer occurred in smokers compared to nonsmokers. If the incidence of lung cancer is the same in both groups, then the relative risk will be 1.0, meaning there is no association. Professor Hill believed that the magnitude of **relative risk should be at least 2.0** to be worthy of suspicion—meaning that smoking should be associated with at least double the risk of developing lung cancer.

In fact, cigarette smoking was associated with an eight to thirty-two times greater increase in risk for developing lung cancer, depending on how heavily people smoked,²⁹ and drinking Broad Street pump water was associated with a fourteen times greater increase in risk for cholera. These are very strong associations! By contrast, most nutrition epidemiology studies report very weak associations between specific foods and specific diseases, with relative risks falling well below the recommended cutoff value of 2.0. In a systematic review of thirty-four epidemiologic studies of diet and heart disease risk, relative risks topped out at only 1.38, with half of all studies coming in at 1.20 or below.³⁰

Commonly, study authors will rephrase a relative risk of 1.2 (which means nothing to most readers) as an “increase in risk of 20 percent”—which readers can more easily understand and makes the risk sound large and important.

Professor Hill’s criteria would have the scientific community dismiss these feeble associations as meaningless, yet the results of nutrition epidemiology studies continue to be published in reputable academic journals and shared via press releases, generating media headlines that everyday people interpret as nutrition advice they can use to make healthier decisions about food for themselves and their families. The public cannot be faulted for making this leap; as Professor Ioannidis affirms: “Authors often use causal language when reporting the findings from [epidemiological studies].... Even when authors add caveats, results are still often presented by the media as causal.”³¹

Nutrition epidemiologists understand that their methodology is only capable of generating hypotheses, so they choose their words carefully

when describing their findings so as not to cross the line between speculation and fact. However, their nuanced rhetoric is easy for journalists to misquote and misinterpret. Even though the authors of our berry study concluded that “women with higher berry intake *appeared* to have delayed cognitive aging by up to 2.5 years [my emphasis],”³² *TIME* magazine reported: “Their findings *confirmed* that women who ate berries at least once a week *were able to* slow down their cognitive decline by about 1.5 to 2.5 years [my emphasis].”³³

Unfortunately, unlike the clear relationship between contaminated water and cholera infections, the relationship between modern diets in all their staggering complexity and chronic diseases like obesity, cancer, and heart disease do not lend themselves well to questionnaire-based epidemiological methodologies.

Nutrition Epidemiology in Media and Policy

Most journalists, clinicians, and lawmakers do not understand the serious shortcomings of nutrition epidemiology, so its unsubstantiated ideas become amplified in media headlines as implicit fact, incorporated into dietary guidelines as policy, and implanted into our collective psyche as gospel. Some of the most influential nutrition documents ever written are grounded largely in nutrition epidemiology, including:

- The U.S. Dietary Guidelines:³⁴ Issued every five years since 1980, this document establishes the diets implemented in all U.S. federal programs (e.g., school lunches and military menus) and sets the tone for nutrition policy for much of the world.
- The World Health Organization 2015 Report on the Carcinogenicity of Consumption of Red and Processed Meat,³⁵ which concluded that processed meat definitely causes colorectal cancer and that red meat probably causes colorectal cancer.
- The 2019 EAT-Lancet report,³⁶ which recommends the near elimination of all animal foods from the human diet.

We will dig into each of these later, but even if you’ve never heard of

these publications, whenever you choose hummus over steak, oatmeal over eggs, or nonfat yogurt over cheddar cheese, you are following their recommendations. But do these dietary choices actually protect or improve your mental or physical health?

Unfortunately, questions such as which foods increase our risk for cancer or which dietary patterns help us live longer lives don't lend themselves to experimentation because they seek to understand risk over very long periods of time—and this is where nutrition epidemiologists, who can repeatedly administer FFQs to large numbers of people over many decades, find a toehold. However, rather than succumbing to the “epidemiology may be flawed, but it's better than nothing” philosophy, why not simply acknowledge that we don't have meaningful scientific ways of addressing these big questions?

The information produced by nutrition epidemiology studies, which forms the lion's share of what we have been taught to believe about food and human health, is wholly untrustworthy. Learning how to identify and avoid nutrition epidemiology findings is therefore essential to protecting your health and the health of those you love.

How to Identify an Epidemiological Study

Whenever you feel disoriented by the fog of any given nutrition science war —whether it's about eggs, red meat, or kale, the secret to keeping your head and finding your way back to safety is learning how to identify and avoid nutrition epidemiology land mines.

The next time you encounter a nutrition science headline, look for the following telltale signs:

- Epidemiology study titles usually include words like “association,” “link,” and “risk.”
- Epidemiology studies tend to be of long duration—years or even decades long.
- Epidemiology studies usually include very large numbers of subjects —thousands or even tens of thousands of people.
- Epidemiology studies often inspire simplistic, appalling, or appealing

headlines such as “Millions of Cardiovascular Deaths Attributed to Not Eating Enough Fruits and Vegetables,”³⁷ “Your Fries May Be Deadly,”³⁸ or “People Who Eat Dark Chocolate Less Likely to Be Depressed.”³⁹ In short, if it sounds too good (or too bad) to be true, it probably is. Who wouldn’t want to believe that all they have to do to ward off depression is eat more chocolate?

WHAT KINDS OF EVIDENCE CAN YOU TRUST?

Epidemiologists study brain food *from the outside in*, by questioning large populations about their dietary habits and looking for patterns in their answers to guess how their food choices affect their mental health. A more logical strategy is to study brain food *from the inside out* by asking what the human brain needs to develop and function properly, and which foods are best at meeting those needs. In our quest for foods that safely and efficiently deliver essential nutrients to the brain, we must set aside the confusing guesswork of nutrition epidemiology, which generates no real data.

Nutrition RCTs generate real data, but their quality and validity depend on how well they are designed, so their findings cannot simply be taken at face value. Clinical case reports and case series can be very illuminating but lack patient controls for comparison, limiting the conclusions we can draw from them.

In short, there is no one fountain of truth we can turn to for answers, so, to gain meaningful insight into human nutrition, we need to gather information from different types of studies and use them to assemble a sturdy, three-dimensional scaffolding of knowledge we can lean on whenever a controversial new headline comes our way. By looking beyond the realm of mainstream nutrition science, asking fresh new questions about tired topics, and seeking areas of agreement where the science is settled, we can more effectively cut through the fog of nutrition science politics and arrive at more enlightened views of brain food.

Fortunately, there is also a wealth of reliable information available for us to draw upon from more rigorous scientific disciplines that are less susceptible to the whims of public nutrition debate, such as anthropology, anatomy, biochemistry, physiology, neuroscience, animal husbandry,

botany, and toxicology. Scientists within these fields have been gathering fascinating facts about living things for generations that are directly relevant to our search for the truth about brain food: human history, plant defense strategies, cell biology, brain chemistry, human digestion, and so much more.

There's no way around it: If it is optimal mental health you seek, you will need to start grounding your food choices in brain biology rather than dietary ideology, and the next two chapters are here to help you do just that. Let's explore the inner workings of your brain, where the secrets to a brain-healthy diet lie waiting to be discovered.

CHAPTER 4

A Guided Tour through Your Brain

You, your joys and your sorrows, your memories and your ambitions, your sense of personal identity and free will, are in fact no more than the behavior of a vast assembly of nerve cells and their associated molecules.

—Sir Francis Crick, *The Astonishing Hypothesis*

Why do you eat?

- You're bored
- You have a craving
- The clock says it's time to eat
- It's a special occasion
- Your grandmother made your favorite dessert
- Your brain (and the rest of your body) needs nutrients

It can be easy to forget this last one, but it's the original and only reason why all living creatures must eat. To build and maintain your cells, you need raw materials (parts) and a fuel source (energy), and these come from macronutrients, micronutrients, oxygen, sunlight, and water.

Macronutrients are versatile molecules we use in *large* amounts: protein, fat, cholesterol, and carbohydrate. All of these can be used as building blocks to construct vital brain components like membranes that surround every cell and keep its contents organized. Macronutrients can also be burned for energy.

Micronutrients are substances we use in *small* amounts to help build, maintain, and energize the brain. These include vitamins (complex

molecules), minerals (simple salts), and essential fatty acids. Like a construction site without workers or tools, without micronutrients, all you'd have is a pile of construction materials and an energy source, with no way to use either one.

In our quest to understand which foods best nourish, protect, and energize the brain, let's embark on a tour of the brain's interior to learn what it's made of and how it works. Neuroscience is complex and can feel overwhelming, but understanding the basics might just inspire a sense of wonder about what your brain cells do all day long and help motivate you to take good care of them. It will also give you the foundation for understanding some of the things that can go wrong inside your brain, and how changing your diet could help. As we explore the fascinating inner world of our most prized organ, keep in mind that absolutely everything we'll encounter along the way comes from food, water, air, and sunlight.

THE CLOISTERED BRAIN

Hidden inside a fortress of bone about a quarter of an inch thick,¹ the brain is the most heavily guarded organ we possess. Fragile and gelatinous, this collection of billions of cells floats in an eight-ounce tank of nourishing *cerebrospinal fluid*² that makes the three-pound brain feel as if it weighs only two ounces.³ Additional layers of security are provided by three protective membranes called *meninges* that surround the brain, tether it in place, and cushion it against traumatic injury.

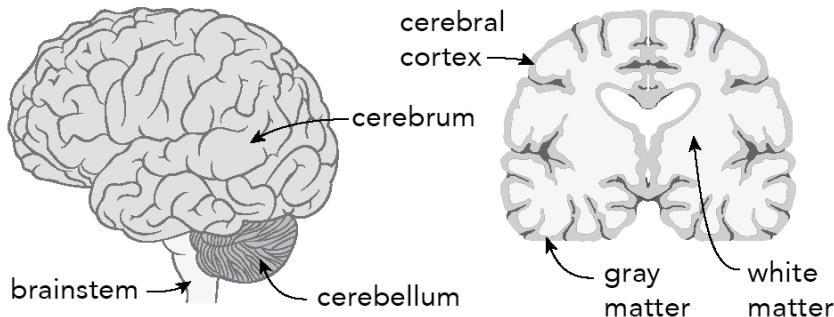
Yet the brain's most advanced defense system is invisible to the naked eye. The microscopic capillaries that crisscross the brain delivering oxygen and nutrients to its hardworking cells are uniquely designed to prevent risky molecules, toxins, and infectious organisms from escaping the bloodstream and invading the brain's sensitive interior. Elsewhere in the body, these minute blood vessels are intentionally leaky to allow easy exchange of nutrients and waste products between the bloodstream and surrounding tissues, but the *endothelial cells* that form the walls of brain capillaries are joined together by interlocking *tight junctions*, creating a leakproof seal. This *blood-brain barrier* is outfitted with intelligent pumps and transport channels that allow it to carefully control which molecules enter and exit the brain's sensitive interior environment.⁴ Situated all along the blood-

brain barrier are transport proteins and receptors designated to escort essential macronutrients and micronutrients into the brain.

A Most Peculiar Place

Before we explore the inner workings of the brain, let's first stand back to appreciate its unusual exterior architecture. The outermost layer of the brain consists of a gray sheet of tissue about one-tenth of an inch thick called the *cerebral cortex* (Latin for “brain bark”). Packed with layers of busy brain cells, the cortex is responsible for our most sophisticated intellectual functions, such as language, reasoning, and planning. The more intelligent a creature is, the larger the surface area of its cerebral cortex must be, which explains the human brain’s strange topography; its crumpled landscape of prominent folds and deep ravines was evolution’s clever way of fitting the vast expanse of our cerebral cortex inside the skull.⁵

Just below this veneer of *gray matter* lies a thick zone of *white matter*—brain cell extensions wrapped in thick layers of *myelin*, an insulating material rich in fat and cholesterol. These cables of white matter connect hubs of gray matter with each other, forming an electrical grid that allows coordinated communication across the entire brain.



Suzanne Smith, brgfx/123rf.com (brain section)

Connecting the brain to the spinal cord is the *brainstem*, an evolutionarily ancient region responsible for controlling basic functions such as breathing and pumping blood. Protruding conspicuously from the rear of the brain is the *cerebellum* (Latin for “little brain”), which coordinates movement and balance. Within the brain’s core lie dense collections of cells that work together to perform specialized tasks. Examples include the *hippocampus*, a seahorse-shaped region where

learning and memory take place (this part of the brain is one of the first to malfunction in Alzheimer’s disease), and the *hypothalamus*, which releases hormones involved in regulating appetite, metabolism, and reproductive cycles.

Deep within the brain lie curiously shaped reservoirs, called *ventricles*, full of cerebrospinal fluid. Cerebrospinal fluid circulates throughout the central nervous system, bathing the brain and the entire length of the spinal cord (this is the same fluid collected during a lumbar puncture or “spinal tap”). This solution, which replenishes itself roughly five times per day,⁶ helps deliver glucose and minerals such as sodium, potassium, and magnesium to the brain, and flushes the brain of toxic waste products and defective molecules that would otherwise accumulate and harm brain cells.⁷ Your brain “washes” itself most effectively overnight during periods of REM (rapid eye movement) sleep when you are dreaming—just one of the many reasons sleep is vital to brain health.⁸

Let’s now turn our attention to the approximately 170 billion individual cells that make up the brain.⁹ Only about half of these are *neurons*,¹⁰ the brain cells famous for conducting electricity; the other half is a diverse collection of *glial cells*, or simply *glia* (Greek for “glue”), which protect and serve neurons.

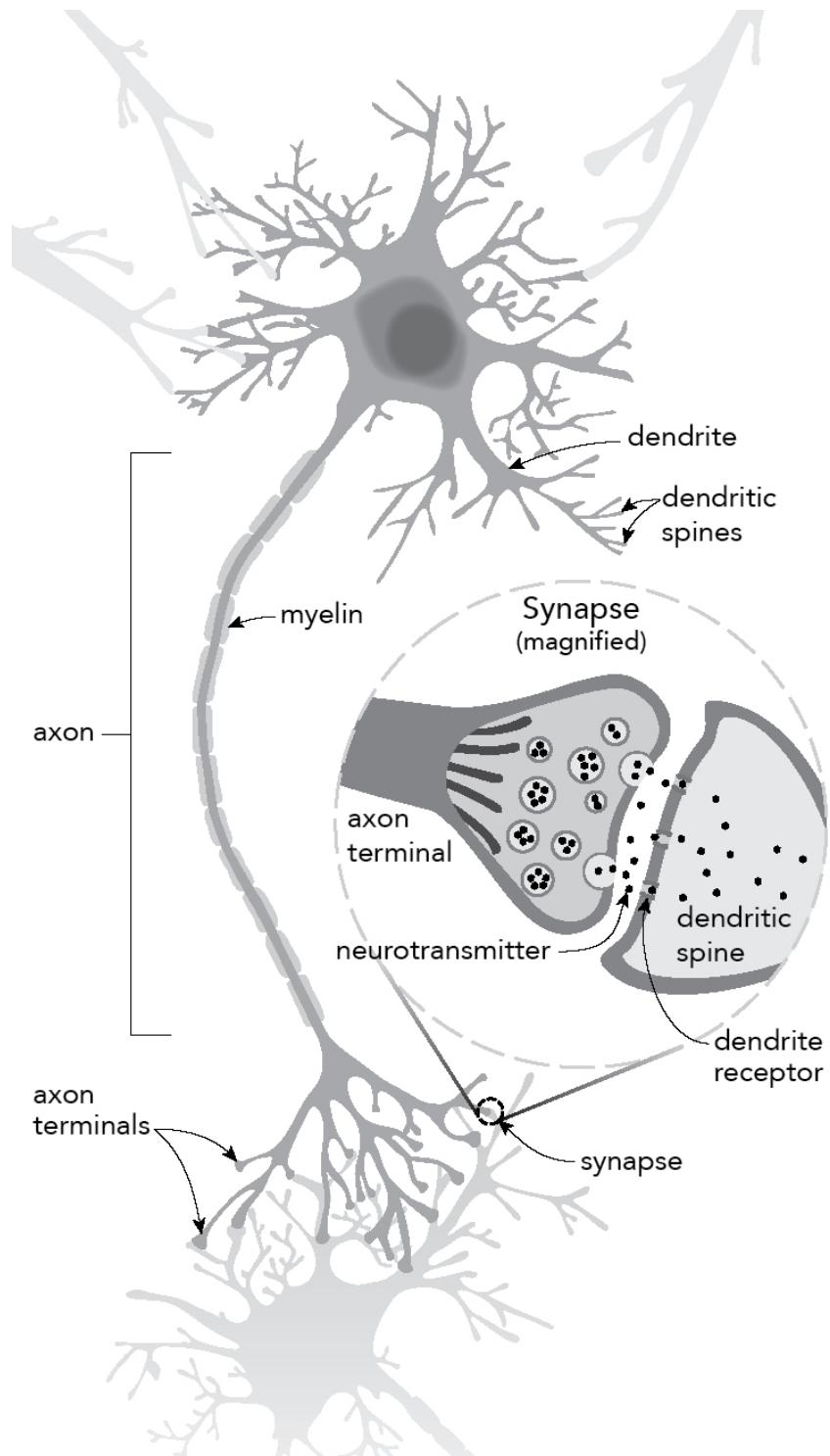
Neurons: Engineered to Conduct Electricity

Your brain’s neurons must be in constant communication with each other and with the rest of your body’s nervous system to smoothly coordinate all of your thoughts, feelings, and behaviors into the cohesive whole that is you. This impressive operating system requires two things: speed and specificity. *Speed* so you can think, move, and react *quickly*—if it takes too long for you to reflexively pull away when you accidentally touch a hot stove, you could suffer serious burns. *Specificity* so you can think, move, and react *appropriately*—after all, how long would you last if you responded to the sensation of touching a hot stove by moving closer to it?

For speed, neurons are engineered to conduct electricity, which allows them to send messages at velocities as high as 200 miles per hour or more.¹¹ For specificity, neurons produce a variety of *neurotransmitters*, such as serotonin and dopamine, capable of delivering different instructions

depending on their type, location, and circumstances.

The receiving ends of neurons feature extensions called *dendrites* studded with *dendritic spines*, tiny docking stations where neurotransmitters from surrounding neurons briefly make contact to drop off their messages. Although a typical neuron only has about six dendrites, it may sport up to 200,000 dendritic spines, allowing it to listen to thousands of neighboring neurons.¹² Depending on the amount and type of neurotransmitter, the receiving neuron may be stimulated enough to fire a jolt of electricity through its cell body and along its *axon* to the *axon terminals* at its tip, where fresh packets of its own neurotransmitters are stored at the ready. When the wave of electrical energy reaches these packets, they break open, releasing neurotransmitters into the gap that separates neurons from each other. These intimate spaces between the axon terminal transmitters of one neuron and the dendrite receivers of the next neuron are called *synapses*.



ANATOMY OF A NEURON

Suzanne Smith

Neurotransmitters attach only momentarily to receptors on the dendrites

of nearby cells to deliver their messages, then as soon as they've accomplished their mission, their parent neuron vacuums them back up, recycling and repackaging them into fresh new storage sacs to wait for the next wave of electricity to set them loose again.

Neurotransmitters

The brain uses dozens of different neurotransmitters; those most familiar and directly relevant to mental health are summarized in the table below. Examples of common medications designed to influence each neurotransmitter are listed in the right-hand column.

Neurotransmitter	Role in the Brain	Medication Examples
Serotonin ¹³	Mood, sleep, sexual desire, anxiety, appetite, temperature regulation, learning, memory	Fluoxetine (Prozac) Sertraline (Zoloft) Escitalopram (Lexapro)
Dopamine ¹⁴	Attention, movement, motivation, learning, memory, reward processing	Bupropion (Wellbutrin) Bethylphenidate (Ritalin)
Norepinephrine ¹⁵	Attention, anxiety, arousal, learning, and memory	Atomoxetine (Strattera)
Acetylcholine ¹⁶	Attention, learning, cognition, memory	Donepezil (Aricept)
Glutamate ¹⁷	Learning, memory, excitation, cell death	Lamotrigine (Lamictal) Memantine (Namenda)
GABA ¹⁸ (gamma-	Cognition, emotion, sleep	Lorazepam (Ativan)

aminobutyric acid)		Clonazepam (Klonopin)
Melatonin	Sleep regulation	

How PSYCHIATRIC MEDICATIONS WORK

Most psychiatric medications are designed to stimulate or suppress the activity of neurotransmitters. A familiar example is the antidepressant family of drugs called *serotonin reuptake inhibitors*, or SRIs (formerly called SSRIs, or selective serotonin reuptake inhibitors), such as fluoxetine (Prozac), sertraline (Zoloft), and escitalopram (Lexapro). One theory about the cause of depression is that serotonin activity is too low. As the name implies, SRIs inhibit the reuptake of serotonin—in other words, they slow down serotonin recycling, which allows serotonin to spend more time in the synapse trying to communicate with other cells. While this strategy sounds logical, it focuses on only one small element of an intricately interconnected system, which may help to explain why medicines targeting serotonin don’t always help as much as we wish they did.¹⁹ For serotonin (or any neurotransmitter) to function properly, the *whole neuron* must be healthy—from the tips of its dendrites to the outer reaches of its axon terminals—and its surrounding environment and neighboring cells must be healthy as well.

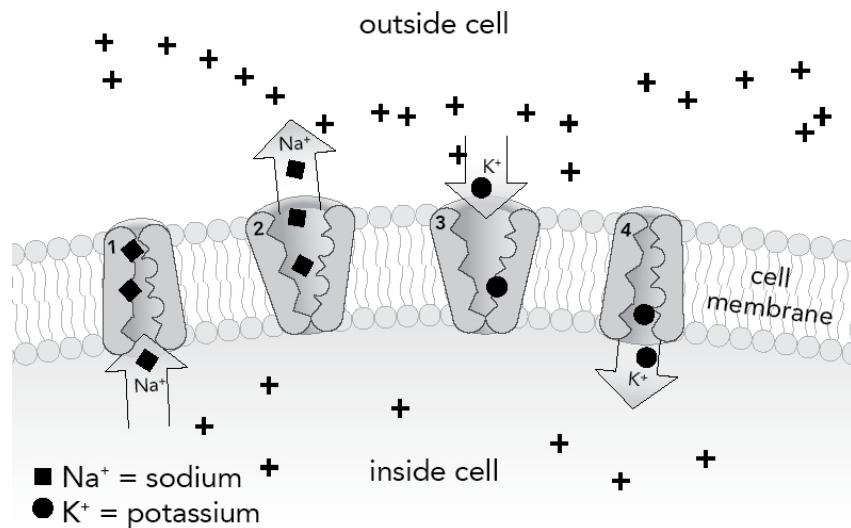
To Fire or Not to Fire

If all of your brain’s neurons were firing all the time, chaos would ensue, so we have elegant systems in place to carefully control their activity. When these regulatory systems malfunction, which is the case in bipolar disorder (and in epilepsy), brain cells might fire when they’re not supposed to, or they might not fire as often as they should. So how does any given neuron in your brain know when to rest and when to fire?

When a neuron releases a neurotransmitter into a synapse, that neurotransmitter then attaches to a receptor on the surface of a neighboring neuron's dendrite and sends a signal to that cell either encouraging it to fire (excitation) or discouraging it from firing (inhibition), depending on the neurotransmitter and the receptor type. The dendrites on the receiving end of a neuron may contain a variety of receptors for a variety of neurotransmitters, but the axon terminals on the transmitting end of a neuron specialize in making, storing, and releasing only one neurotransmitter. It's as if each neuron can understand an entire vocabulary but only knows how to say one particular word. This multiple-input, single-output system allows each neuron to weigh the opinions of thousands of its neighbors before deciding whether or not to fire.

The electricity generated by a firing neuron is made possible by essential minerals that come from the foods you eat and the water you drink. Important examples include sodium, potassium, and calcium, which are all positively charged electrical particles. The chemistry term for these charged salt particles is *ions*, and the everyday clinical term for these is *electrolytes*.

A neuron "at rest" is anything but relaxed. Thanks to hardworking sodium-potassium pumps embedded along the length of its axon's membrane, it's quietly brimming with electrical potential. Left to their own devices, sodium and potassium ions would simply distribute themselves uniformly within and around the axon until the concentration of sodium and potassium inside and outside the cell became equal. The pumps create an electrical imbalance between these two minerals by forcing lopsided exchanges of sodium for potassium; for every three sodium ions pumped out, only two potassium ions are pumped in, until the inside of the axon is negatively charged compared to the outside. Sodium and potassium would love to equalize their concentrations and relieve the electrical tension, but the only way they can cross the membrane is through special channels which are usually closed, trapping sodium and potassium in a high-energy state of imbalance.

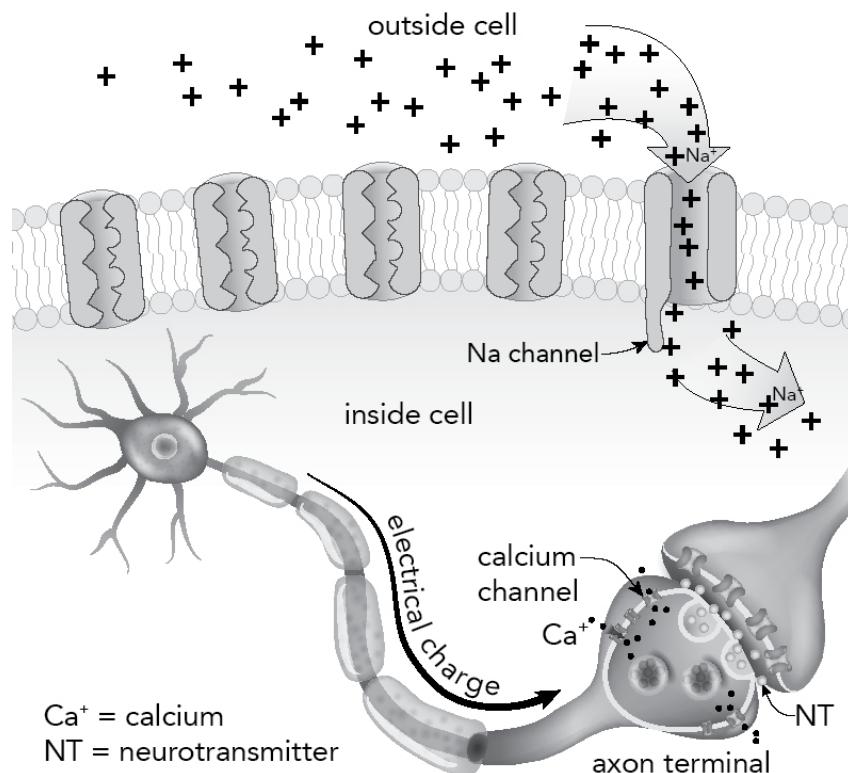


SODIUM-POTASSIUM PUMPS IN A RESTING NEURON

The sodium-potassium pumps in a neuron's axon membrane maintain an electrical imbalance by pumping three sodium molecules outside the cell for every two potassium molecules it pumps into the cell.

designua/123rf.com, Suzanne Smith

Like a slingshot stretched to its maximum length, the “resting” neuron sits primed and ready to fire at a moment’s notice. Only if incoming neurotransmitter signals are strong enough to excite the cell to fire will sodium channels fly open and allow sodium ions to rush back into the cell. This wave of positive electrical charge created by incoming sodium particles zips all the way down the axon to its terminal tips, where it forces calcium channels open. Calcium then storms into the axon terminal, triggering neurotransmitter storage pouches to burst open and release their contents into the synapse to stimulate the next neuron in the chain.



FIRING NEURON

A sodium channel embedded in the membrane of the neuron's axon opens to allow sodium ions that had been forcibly removed from the cell to come rushing back in (top), stimulating an electrical current that travels to the axon terminal where it stimulates the release of neurotransmitters into the synapse (bottom).

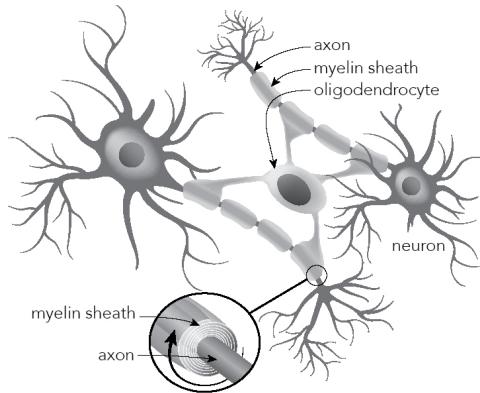
Suzanne Smith, designua/123rf.com (sodium-potassium pump); guniita/123rf.com (neuron)

As you might imagine, this exquisite electrochemical cycle, which is taking place in tens of billions of neurons as often as three hundred or more times per second²⁰ demands a tremendous amount of energy and a steady supply of nutrients, and the electricity it generates must be controlled to prevent the brain from short-circuiting. This is why behind every flashy electrical neuron there are trusty glial cells.

These under-appreciated caretaker cells come in four varieties, each devoted to supporting high-maintenance neurons in its own way.

Oligodendrocytes: These octopus-like cells sprout long membrane arms that wind themselves around the axons of nearby neurons—as many as one hundred times—like a roll of tape, insulating them in a thick, fatty coating

of myelin. A single oligodendrocyte can produce up to eighty myelin coils.²¹ It is myelin that makes white matter appear white, and that enables electrical signals to race swiftly and efficiently through the brain and the rest of the nervous system. The importance of myelin is made plain in multiple sclerosis, an autoimmune disease in which the body attacks and destroys myelin sheaths. Axons laid bare by multiple sclerosis are vulnerable to damage and can't conduct electricity properly. Brain symptoms of this devastating disease can include slurred speech, double vision, depression, and psychosis.²²



OLIGODENDROCYTE

Oligodendrocytes wrap neighboring axons with myelin.
designua/123rf.com (oligodendrocyte), guniita/123rf.com (myelin)

Astrocytes are star-shaped guardians of synapses. Synapses are where neurotransmitters are manufactured, so they have a voracious appetite for amino acids, vitamins, and minerals. Like attentive parents, astrocytes feed them, fuel them, and clean up after them, ensuring that these bustling cellular intersections always have everything they need in just the right amount.²³

Microglia, the immune cells of the brain, are branched cells that vigilantly patrol their local neighborhood, keeping things tidy and looking for threats. If one of their many probing fingers encounters an invader such as a virus, the entire cell will instantaneously morph into a Pac-Man-like configuration, swallow the virus whole, and then revert to its original shape.²⁴ Microglia play a major role in brain inflammation, one of the root causes of psychiatric conditions.

Ependymal cells line the walls of ventricles, continuously producing

fresh cerebrospinal fluid and encouraging it to circulate by waving long extensions of their bodies called *cilia* back and forth.

Now that you've been introduced to the brain's outer contours and its microscopic structure, it's time to go down to the molecular level to discover which macronutrients and micronutrients you need in order to nourish, protect, and energize this magnificent organ.

YOUR BRAIN IS MADE OF MACRONUTRIENTS

Protein, fat, carbohydrate, and cholesterol are macronutrients—versatile molecules we use in large amounts either as building blocks or fuel sources. All four macronutrients are made of carbon, hydrogen, and oxygen, but protein is unique in that it also contains nitrogen. The body can turn fat into carbohydrate or carbohydrate into fat, but it can't turn fat or carbohydrate into protein. This explains why you can survive eating a very low-fat diet or a very low-carbohydrate diet, but you cannot survive for long eating a low-protein diet.

Protein (Amino Acids)

Protein is the most important macronutrient in our diet. It is often said that we must eat protein regularly because it is the only macronutrient we can't make or store, but that's not quite right. While it's true we have no way to store it, we do make all of our own proteins—out of amino acids. So, we don't eat protein for the proteins themselves; what we're after is the amino acids they contain. Case in point: no matter how much calamari you consume, you will never be able to sprout a suction cup; instead, you break the proteins in calamari down into individual amino acids and rearrange them into the human proteins you need to build a hair follicle, a hormone, or a hamstring.

Just as we can create thousands of different English words using only twenty-six letters, we can build thousands of different proteins using only twenty-two amino acids.²⁵ Nine of these amino acids are essential (impossible to make ourselves), eight are conditionally essential (we can't always make enough to meet our needs), and the remaining five are nonessential (we make them ourselves).

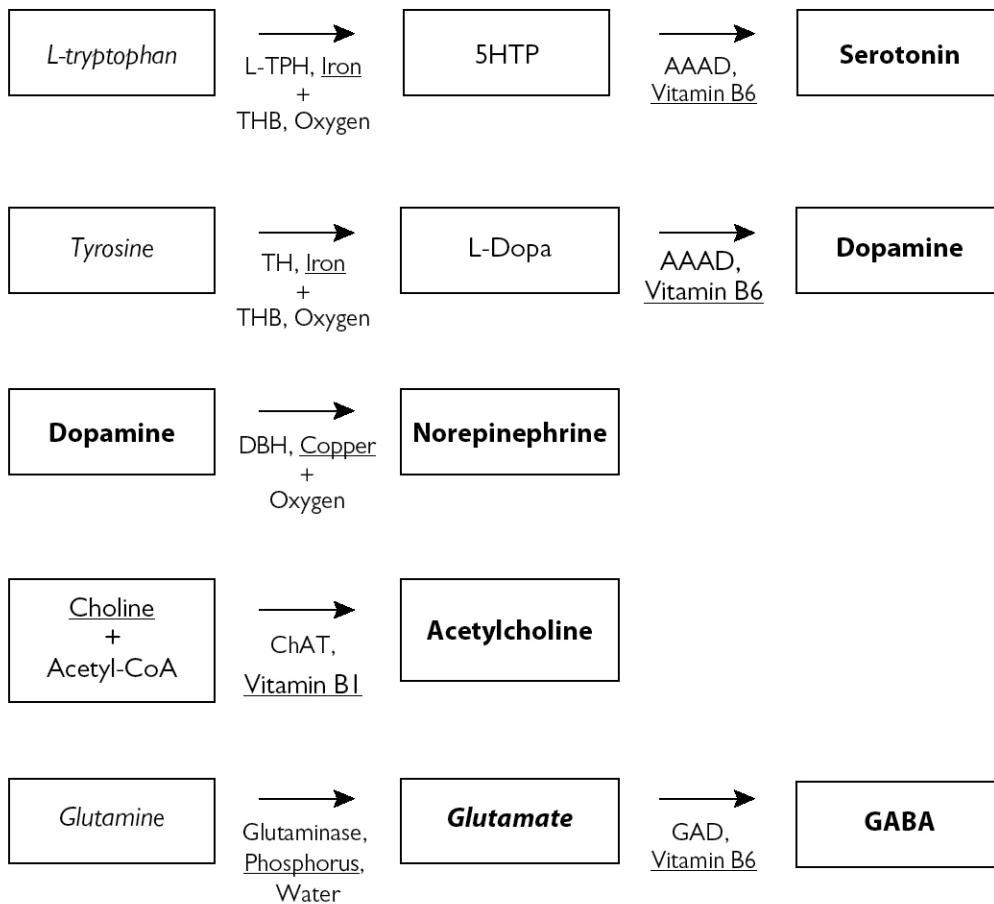
Essential	Conditionally Essential	Nonessential
Histidine	Arginine	Alanine
Isoleucine	Cysteine	Asparagine
Leucine	Glutamine	Aspartate
Lysine	Glycine	Glutamate
Methionine	Proline	Serine
Phenylalanine	Selenocysteine	
Threonine	Tyrosine	
Tryptophan	Taurine ²⁶	
Valine		

We must eat all nine essential amino acids regularly, and we must also eat the eight conditionally essential amino acids when protein requirements are higher, including times of growth, stress, injury, illness, and during pregnancy and breastfeeding.

Like all organs, the brain uses amino acids for a multitude of purposes. For example, as you can see from the ingredient lists in the following sidebar, most neurotransmitters are made from amino acids. The enzymes needed to build those neurotransmitters are also made of amino acids, as are the receptors that receive their messages.

THE NUTRITION-NEUROTRANSMITTER CONNECTION

Neurons transform amino acids and other nutrients into neurotransmitters using chemical reactions that involve enzymes, vitamins, and minerals, so if you are short on certain nutrients, your neurons will not be able to manufacture all the neurotransmitters your brain needs to function properly. Notice, for example, how important iron and vitamin B6 are to neurotransmitter construction. If you have iron deficiency, you don't just have a blood problem, you have a brain problem.



Legend: **neurotransmitter**, **amino acid**, **vitamin/mineral**

Enzyme abbreviations: L-tryptophan hydroxylase (L-TPH), tetrahydrobiopterin (THB), aromatic L-amino acid decarboxylase (AAAD), Tyrosine hydroxylase (TH), dopamine beta-hydroxylase (DBH), choline acetyltransferase (ChAT), acetyl coenzyme A (Acetyl-CoA)

The ideal daily requirement for protein remains a topic of debate in nutrition science circles, with most sources recommending a *minimum* of between 0.4 to 0.6 grams per pound ideal body weight (0.8 to 1.2 grams of protein per kilogram ideal body weight) per day. I'll help you estimate your daily requirement in [chapter 17](#), but to give you a sense of how broad the range can be: if your ideal body weight is 140 pounds, many experts would recommend you eat at least 56 grams of protein per day (equivalent to about ten ounces of salmon) while others would recommend at least 84 grams per

day (equivalent to about fifteen ounces of salmon). Amino acid deficiencies are unlikely so long as you have access to adequate amounts of *high-quality* protein, meaning foods that contain a healthy balance of these amino acids. As we'll explore in more detail later, nearly all animal proteins contain the full complement of amino acids whereas some plant proteins are low in certain amino acids, most commonly methionine, cysteine, and lysine.²⁷

Fat (Fatty Acids)

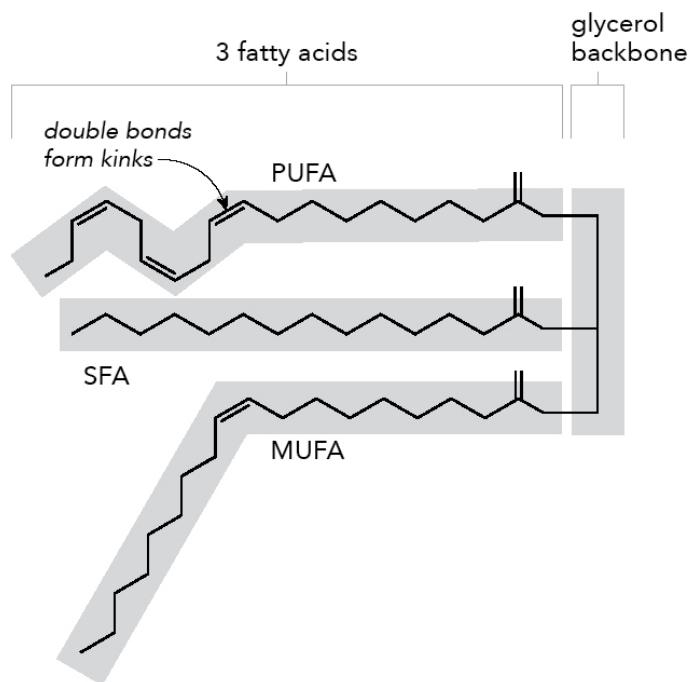
Just as protein molecules are made of individual amino acids, fat molecules are made of individual fatty acids. Fat molecules are also called *triglycerides* because each one contains three fatty acids attached to a simple sugar backbone called *glycerol*, like three flags flying on a flagpole. (Standard cholesterol blood tests include a test for triglycerides, which is a measure of how much fat is traveling through your bloodstream.) Fatty acids come in a variety of lengths (short, medium, or long) and types (saturated, monounsaturated, or polyunsaturated).

- **Saturated** fatty acids are straight carbon chains “saturated” with the maximum possible number of hydrogen atoms attached to carbon atoms by strong “single” bonds. Saturated fats are very stable and tend to be solid at room temperature. Coconut oil and butter are familiar examples of fats high in saturated fatty acids.
- **Monounsaturated** fatty acids (or MUFAs) are bent in one spot where a fragile carbon-carbon double bond replaces strong carbon-hydrogen single bonds. This weak link causes MUFAAs to melt more easily. MUFAAs are liquid at room temperature, so we call them oils. Olive oil and avocado oil are high in MUFAAs.
- **Polyunsaturated** fatty acids (or PUFAs) have even fewer hydrogens and contain multiple double bonds, therefore they contain multiple kinks. These oils are unstable and go rancid easily. Many nuts, seeds, and oily fish are naturally high in polyunsaturated fatty acids.

Each of these fats plays important roles in our bodies, so it is incorrect to

think of unsaturated fats as inherently healthy and saturated fats as inherently unhealthy. In fact, the majority of the fat in our bodies is saturated fat, by design. Some of it is monounsaturated, but only a small percentage of it is supposed to be polyunsaturated.

The liquid properties of unsaturated fats make them important ingredients in body fluids such as those that lubricate our eyes (tears) and joints (synovial fluid), whereas sturdier, denser saturated fats insulate us against the cold, cushion our internal organs against damage, and serve as a lightweight, compact, flexible way to store energy. Unlike plants, which store most of their energy as starch (think yams and turnips), we animals need to move about in the world and can't afford to be weighed down by unsightly potato-like structures. Fat also holds more than twice as much energy per gram as starch can, which is fortunate, because hardworking muscles, hearts, and brains use a lot more energy than sedentary stems, leaves, and flowers. We have very limited capacity to store carbohydrate as starch, so the liver converts any surplus carbohydrate we eat into saturated fat for easy storage. This bears repeating: if you eat more carbohydrate than you can burn right away or store as starch, your liver will turn it into saturated fat, not unsaturated fat, because saturated fat is the most compact and practical way to store energy. It stands to reason that if saturated fat were inherently bad for us, the body wouldn't be designed to do this. If you were to try to store energy as unsaturated fat, you would sag everywhere and begin to resemble a Shar-Pei.



TRIGLYCERIDE STRUCTURE

Triglycerides are composed of three fatty acids attached to a glycerol backbone.

This illustration includes the saturated fatty acid (SFA) myristic acid, the monounsaturated fatty acid (MUFA) oleic acid, the primary fat in olive oil, and the polyunsaturated fatty acid (PUFA) alpha-linolenic acid (ALA), an omega-3 fatty acid.

Suzanne Smith

Your brain is the fattiest organ in your body (other than fat tissue itself). If you could wring out all of its water, you'd be left with a wad of material containing about fifty percent fat.²⁸ Why is the brain so fat? In a word, membranes. Membranes, which consist of two layers of fat, are flexible barriers that intelligently organize our bodies into individual compartments. Every one of your cells, and even many of the smaller structures inside your cells, are wrapped in membranes. Membranes contain a mixture of saturated fat (for structural integrity) and unsaturated fat (for fluidity). The brain is particularly rich in fat compared to other organs primarily because the myelin required to insulate brain circuitry is nothing more than tightly coiled membranes—and the brain's white matter contains more than 60,000 miles of myelinated axons.²⁹

Roughly speaking, about one-third of brain fat is saturated fat (palmitic and stearic acid), and another one-third of it is a monounsaturated fat called

oleic acid. Oleic acid is important to brain health, including for the creation of new memory circuits,³⁰ but it is not an essential fatty acid, because brain cells can make it from other molecules. In fact, your brain is capable of building all of these fats from scratch out of simple glucose molecules, but it generally prefers to start with pre-assembled fatty acids, which it absorbs from the bloodstream.³¹ The remaining one-third of the fat in your brain consists primarily of two remarkable polyunsaturated fatty acids: an omega-6 fatty acid called *arachidonic acid* and an omega-3 fatty acid called *docosahexaenoic acid* (DHA). Far from simple chains of stored energy, these elongated PUFAs possess unique properties that make them essential to the brain's specialized electrical functions.

Arachidonic acid is a multitalented molecule with four double bonds that force it into an unusual hairpin shape. It is required for brain development, membrane flexibility, cell signaling, and immune system function.

DHA's six double bonds are arranged in a unique configuration that gifts it with quantum mechanical properties.³² More simply stated, DHA acts as a semiconductor or buffer of electricity, so we find it in electrical hotspots around the brain: synapses where electrochemical signals are being converted into memories, the retina of the eye where sunlight is transformed into electricity, and the electron transport chain where food molecules are turned into energy (more about this later). As distinguished Imperial College brain-lipid scientist Professor Michael Crawford writes, DHA is “essential for visual acuity and the truthful execution of the neural pathways which make up our recollections, information processing, and consciousness.”³³ DHA also directs the organization of the cerebral cortex, meaning that a deficiency of this precious fatty acid in early life could have profound and irreversible effects on many aspects of a child’s intelligence, including critical reasoning skills, language, and learning capacity.

One of the many services DHA and arachidonic acid perform in the brain is to generate first-responder molecules for the brain’s immune system. These fatty acids are firmly embedded within cell membranes, where they wait patiently until the immune system calls them up for duty. If the brain is under threat and needs to mount an inflammatory response, enzymes will free arachidonic acid from the membrane and convert it into a cascade of molecules that *promote* inflammation. When the time comes for

healing, enzymes will free DHA from the membrane and convert it into a cascade of molecules that *resolve* inflammation.³⁴

There is one more omega-3 PUFA that deserves special mention, even though the brain itself contains very little of it: eicosapentaenoic acid (EPA). Just as DHA promotes healing in the brain, EPA promotes healing in the rest of the body.³⁵

Our bodies can assemble most of the fats they need out of glucose or out of other individual fatty acids, but not EPA, DHA, and arachidonic acid. We don't possess the enzymes needed to make these special long-chain PUFAs from scratch, so we would be wise to eat foods that already contain them—and they are only found in animal foods. A far less reliable option would be to consume other long-chain PUFAs (namely *linoleic acid* and *alpha-linolenic acid*, both of which are found in plant fats as well as animal fats) and try to convert them into the PUFAs we need, but as we'll discuss later, not only is our capacity to do this extremely limited, but consuming more than very small amounts of linoleic acid is risky.

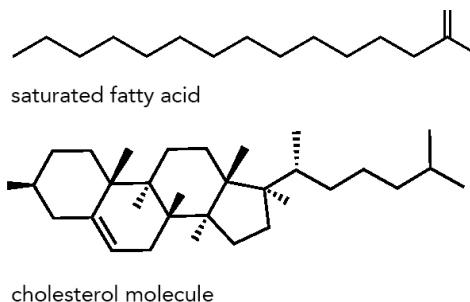
Vitamins A, D, E, K1, and K2 are the “fat-soluble” vitamins, which means we can't absorb them unless we consume them with some fat. The standard recommendation is to obtain 20 percent to 35 percent of our daily calories from fat,³⁶ and we are often advised to choose monounsaturated and polyunsaturated fats over saturated fats—a recommendation we will challenge throughout this book. There is broad agreement that we require a source of essential PUFAs and that we must consume enough fat to absorb fat-soluble vitamins. Beyond these two undisputed recommendations, there is little agreement about the quantity and type of dietary fat humans should ideally eat, but as we'll see, there can be advantages to higher-fat diets, particularly where the brain is concerned.

Cholesterol

Most people view a high-cholesterol meal as a heart attack on a plate, and most physicians think of cholesterol in the blood as something to be minimized with medication. But what exactly is it?

Cholesterol is a hard, waxy substance essential for all animal life. We can't build hormones like estrogen, testosterone, or vitamin D without cholesterol, and its mandatory presence in our cell membranes should be

comforting to us all, as its sturdy constitution contributes firmness and helps maintain their structural integrity. People often utter the words “fat” and “cholesterol” in the same breath, but not only do they look nothing alike, they serve very different purposes.



Notice that fat is a humdrum chain, whereas cholesterol sports a fancy honeycomb structure made of three hexagons plus a pentagon (in medical school we affectionately called it “three rooms and a bath”). Our cells can make a triglyceride (fat) molecule in just eleven steps, whereas it takes more than thirty chemical reactions to hammer together a single cholesterol molecule. Saturated fat is easily chopped up and burned for energy, but cholesterol is indestructible. The only way to dispose of excess cholesterol is to send it to the liver to be turned into bile and excreted by the bowel.³⁷

Although the brain represents only 2 percent of your total body weight, it contains 20 percent of your body’s cholesterol—more cholesterol than any other organ in your body. What’s all that cholesterol doing up there? Once again, the answer is membranes. A full seventy-five percent of your brain’s cholesterol is wound up in myelin, and the rest resides in the membranes that line neurons and glial cells. In brain synapses, cholesterol plays an important role in cell-to-cell communication by helping to form *lipid rafts*—stiffened zones within cell membranes that stabilize the signaling proteins needed for neurotransmitter interactions.³⁸

Cholesterol is too big and bulky to enter the brain across the blood-brain barrier, so not a single molecule of your brain’s cholesterol comes from the cholesterol in your food. Instead, the brain makes all of its own cholesterol on-site, from scratch. I ask you: Why would the brain go out of its way to intentionally produce large quantities of a substance that’s bad for you? It wouldn’t. It’s really smart. It’s a brain.

Once the brain has gone to the trouble of building cholesterol, it tends to

hold on to it for a long time; cholesterol molecules inside the brain can last for years. And brain cells aren't the only cells capable of making their own cholesterol; in fact, every cell in the human body (except red blood cells) is equipped to do this. As for whether you can eat too much cholesterol, we'll explore that question in [chapter 11](#) when we discuss eggs.

Carbohydrate

Much like fat, the sugars and starches that make up the carbohydrate family are simple, versatile molecules that can easily be chopped up and burned for energy or combined with other molecules to build cell components. The two most important carbohydrates in our bodies are glucose (a simple sugar) and *glycogen* (a complex starch).

Our bodies digest all of the sugars and starches we eat—whether they come from flour, fruit, or potatoes—down to glucose, a simple sugar ring that circulates in the bloodstream to supply cells throughout the body with energy. Certain organs, especially the liver and muscles, can store some glucose by linking tens of thousands of glucose molecules together into glycogen and squirreling it away for future use.

You must have some glucose circulating in the blood at all times to feed your brain and other organs (the brain can only store a tiny quantity of glycogen), but that glucose does not need to come from your diet, because your liver can make all the glucose your bloodstream needs out of fatty acids (and certain amino acids if need be), via a process called *gluconeogenesis*—literally, “new glucose formation.” This means that *eating carbohydrate is entirely optional*. We’ll cover carbohydrate in more detail in the coming chapters.

I hope you’ve enjoyed our tour of brain anatomy. By seeing firsthand what the brain is made of, we can begin to assemble our brain-healthy diet ingredient list:

- The brain needs high quality protein to provide it with the full complement of essential amino acids such as tryptophan, which it uses to build serotonin, and tyrosine, which it uses to build dopamine.
- The brain makes its own cholesterol and can even make its own

saturated fats and monounsaturated fats if need be, but it must have access to reliable sources of two polyunsaturated fatty acids: DHA (an omega-3 fatty acid) and arachidonic acid (an omega-6 fatty acid).

- The brain requires all essential micronutrients, with vitamin B1, vitamin B6, iron, choline, and copper being most directly involved in neurotransmitter production (vitamins B9 and B12 support this process indirectly).
- The brain requires electrolytes (mineral salts) to conduct electricity, with sodium, potassium, and chloride being most directly involved.

For your brain to function at its best, it must be healthy through and through, which starts by nourishing it properly. If even one essential amino acid is in short supply, you won't have the ingredients required to build all of the vital molecules it needs like neurotransmitters, receptors, or calcium channels. Without the right fats in the right proportions, brain development could go awry, membranes may become too flimsy or too rigid, and the brain's immune system could malfunction. Now that we know more about what it takes to nourish the brain's infrastructure, it's time to move on to the next pillar of robust mental health: brain energy production.

CHAPTER 5

The Magic of Brain Metabolism

Welcome to the human brain, the cathedral of complexity.

—Peter Coveney and Roger Highfield, *Frontiers of Complexity*

The brain demands about twenty percent of your body's total daily energy supply—ten times what you would expect for an organ of its size. This voracious appetite for fuel is due largely to its sodium-potassium pumps, which work diligently around the clock to force charged salt particles apart and maintain the electrical imbalance needed to prime neurons to fire.

To supply your brain with energy, you need fuel, of course, but you also need micronutrients like B vitamins and iron to help operate the cellular machinery that burns that fuel. Required in minuscule amounts, micronutrients play an outsized role in cellular life. In the dizzying array of intersecting burning and building pathways that make up the miracle of brain metabolism, vitamins and minerals are there at every turn, activating enzymes, influencing genes, conducting electricity, and performing hundreds of other metabolic chores.

Brain energy production begins in the gut, where your stomach, intestines, liver, and pancreas cooperate to break down the foods you eat into small molecules to fuel your brain. So, rather than diving headfirst into brain metabolism, let's start with gut metabolism and work our way up to the brain so you can see how each system behaves when you are eating and when you are fasting. I've highlighted certain nutrients along the way, but in appendix C you'll find a complete list of essential micronutrients, along with a description of the key roles each one plays in energizing and supporting your brain.

Insulin as Metabolic Maestro

The word “metabolism” refers to the symphony of chemical reactions that cells use to extract energy from food and conduct their daily business. Metabolic reactions fall into two categories: construction (*anabolic*) pathways involved in building, growth, and storage projects, and destruction (*catabolic*) pathways involved in burning, recycling, and demolition projects. These opposing forces are tightly controlled by an elegant system of enzymes, sensors, feedback loops, and hormones that determine how active these pathways should be at any given time. (Hormones are chemical messengers produced by glands and released into the bloodstream in tiny amounts to influence the behavior of faraway cells.) Many hormones work in concert to regulate your metabolism, but one of the most important ones—and arguably the one you have the most control over—is insulin.

Deep within your abdomen, your pancreas is continuously releasing insulin into your bloodstream to support your metabolism, and the amount of insulin your pancreas produces at any given time is largely determined by your diet. All foods (and most beverages) stimulate the pancreas to produce some amount of insulin, but as a general rule:

- carbohydrates cause the greatest rise in insulin levels
- most proteins cause a mild to moderate rise in insulin
- fat barely stimulates insulin at all

The most powerful driver of insulin production is rising blood glucose, and the most powerful driver of rising blood glucose is dietary carbohydrate (sugars and starches)¹—with refined carbohydrates such as sugar, fruit juice, and processed grain products (flour, white rice, instant oats, breakfast cereals, etc.) demanding the greatest amount of insulin. (Remember, starch is just lots of glucose molecules linked together, so foods don’t have to be sweet to raise your blood glucose.)

When your pancreas senses that glucose levels are rising, it releases insulin into the circulation to squirrel the extra glucose away into cells and bring blood glucose levels back down again. Insulin’s ability to keep glucose in check is very important because high blood glucose is toxic and

will slowly damage every cell in the body. People with diabetes inject themselves with insulin to control high blood glucose levels, so it's common to think of insulin as a simple blood glucose regulator, but the whole truth is that insulin is a *master growth hormone*—a chief orchestrator of anabolic metabolism. Put simply, insulin tells your cells what to do with incoming food molecules. Virtually every cell in the brain and the rest of the body wears insulin receptors on its surface so it can listen for insulin's cues. Like musicians focused on a conductor's baton, your cells constantly fine-tune their behavior in response to insulin as its blood levels rise and fall.

Insulin High? Burn Glucose, Make Fat

When insulin is high and glucose is abundant, such as after a typical balanced meal that contains a decent amount of carbohydrate, your metabolism shifts into growth and storage mode. You burn glucose for energy and turn excess calories into fat.

Insulin is the hormone of plenty, announcing to cells far and wide that a fresh supply of nutrients is available for growth, repair, and storage projects. In many cases, insulin even micromanages the delivery of glucose to individual cells. For example, when insulin attaches to insulin receptors on muscle and fat cells, those cells respond by opening chutes that allow glucose molecules to tumble in.² Insulin then directs those cells to fire up their assembly lines so they can use incoming nutrients to build new molecules. To fuel all of this activity, insulin throws glucose into the cell's metabolic furnaces to chop it up and turn it into energy. If there is more glucose in your bloodstream than your cells can use right away, insulin instructs muscle and liver cells to knit those surplus glucose molecules together into glycogen (starch) to top off their energy reserves, a process that requires vitamin B7. Once those starch silos are full, insulin instructs the liver to turn the rest of the glucose into triglycerides (fat) and ship them out to fat cells for storage.

Insulin Low? Burn Fat, Make Glucose

When insulin is low and glucose is limited, such as when you are fasting or eating a ketogenic diet, your metabolism shifts into fat-burning mode and

you make your own glucose.

The combination of low insulin and dipping glucose triggers fat cells to break down stored fat molecules into fatty acids (which requires vitamin B5) and release them into the circulation to feed hungry cells.³ If your metabolism is healthy and flexible, most of your cells' activities will readily switch from burning glucose to burning fatty acids. To serve the needs of the few systems that require glucose at all times, the pancreas releases a hormone called *glucagon*, which orders the liver (aided by vitamin B6) to break down its stored glycogen into glucose molecules and release them into the bloodstream to maintain adequate blood glucose levels. However, since the liver can only store about 400 calories' worth of glycogen,⁴ glucagon also turns on the liver's gluconeogenesis pathways (which use vitamins B6 and B7) to build brand new glucose molecules out of fat and protein. Whereas we can only store *hours'* worth of energy as glycogen, we can store *months'* worth of energy as fat, allowing us to energize ourselves for very long periods of time between meals if necessary.

Your cells' ability to switch most of their metabolic activities back and forth between glucose and fat ensures that they get their energy needs met around the clock regardless of how much carbohydrate you eat or how often. If we'd needed constant access to carbohydrates, our species wouldn't have lasted very long. Indeed, your cells *expect* you to spend time in a low-insulin "fat-burning mode" on a regular basis. Only when you are vigorously burning fat can your liver generate ketones to help energize your brain,⁵ and there are distinct advantages to this metabolic state.

When you are vigorously burning fat, there will be more fatty acids entering your liver than it can use for energy. You can't store the extra fatty acids as fat, because insulin is low, so your fat cells are in burning mode, not in storage mode—*your fat storage pathways are turned off*. Instead, the liver takes those surplus fatty acids (which can be fairly long molecules) and breaks them down into small fragments called *ketone bodies* (aka ketones) that are faster and easier to burn. By turning excess fatty acids into ketones, your liver is essentially "pre-digesting" them for your busiest organs, most importantly your heart, muscles, kidneys, and brain—organs that need access to fast energy and don't have time to spend tediously chopping up long molecules. In fat-burning mode, heart and muscle cells have the luxury of choice: they can burn fatty acids, ketones, or their own

supplies of stored glycogen (which they selfishly do not share with the rest of the body) for energy. In contrast, the brain can only store tiny amounts of glycogen,⁶ and its neurons can't burn fatty acids,⁷ so under lower-glucose conditions, the brain becomes dependent on ketones to help meet its high energy demands.

KETONE CHEMISTRY

Ketone bodies come in three forms: acetoacetate (AcAc), acetone, and beta-hydroxybutyrate (BHB). Acetoacetate is the only one cells can burn, but it isn't very stable, so it doesn't travel well. As AcAc circulates in the bloodstream, some of it is lost in the urine and some spontaneously breaks down into acetone, a waste product that is exhaled in the breath. To minimize this loss of precious energy molecules, the liver turns as much AcAc as possible into BHB for transport, because BHB is stable in the bloodstream.⁸ BHB travels well but cells can't burn it, so once BHB has safely arrived at its destination, cells turn it back into AcAc and then break that down for energy. (We'll refer to all three of these molecules as "ketones" even though BHB isn't technically a ketone at all; it's an organic acid.)

BRAIN METABOLISM

University of Sherbrooke brain metabolism expert Professor Stephen Cunnane describes the brain as a hybrid engine that ideally runs on a mixture of glucose and ketones.⁹ This dual-fuel system ensures that your brain has a constant supply of energy, regardless of what you eat and how often.

Your Brain on Glucose

The brain continuously sips glucose from the bloodstream through one-way

valves stationed along the blood-brain barrier. This system keeps the brain's glucose level about 80 percent lower than the blood glucose level,¹⁰ so if your blood glucose is 100 mg/dl, your brain glucose will be about 20 mg/dl; if your blood glucose is 200 mg/dl, your brain glucose will be about 40 mg/dl, and so on, so *the higher your blood glucose, the higher your brain glucose.*

Inside the brain, hungry neurons siphon up about eighty-five percent¹¹ of those incoming glucose molecules¹² but since they can't burn glucose as well without insulin, the blood-brain barrier is studded with insulin receptors that escort insulin into the brain. Like a foreman arriving at a job site, when insulin attaches to the surface of a neuron, it flips on a series of metabolic switches inside the neuron to fire up glucose processing, and production lines immediately begin humming with activity, burning glucose for energy and building new molecules.

Your Brain on Ketones (Plus Glucose)

Whenever blood glucose and insulin levels fall, blood ketone levels will rise. As they circulate through the brain, transporters along the blood-brain barrier welcome them inside, so, just as with glucose, *the higher your blood ketones, the higher your brain ketones,*¹³ and just as with glucose, neurons display receptors on their surfaces to capture those ketones so they can burn them for energy. However, your brain cannot run on ketones alone (we'll see why shortly), so, even when you're vigorously burning fat and your blood ketones are relatively high, your liver continues to produce glucose to support brain operations that require glucose.

Unless something is terribly wrong, there is always plenty of glucose in your bloodstream, but ketones can be harder to come by. In most well-fed people who eat a typical diet, ketone levels tend to be extremely low, because high-carbohydrate diets keep insulin levels too high to allow significant ketone production to occur. Ketones typically only begin rising to meaningful levels if you're sleeping, fasting, exercising, restricting calories, or eating a ketogenic diet. Under those conditions, your body will burn more fat, and your liver will turn some of that fat into ketones. In other words, you will enter a state of *ketosis*.

When insulin is high and ketones are low, your brain runs almost

exclusively on glucose. As insulin falls and ketones rise, your brain begins burning more ketones and less glucose. Most people think of ketones as an emergency backup fuel for the brain, but given the option, the brain will continue to absorb and burn ketones, even when there's plenty of glucose available.¹⁴ The brain's voluntary refusal to run entirely on glucose when ketones are present is intriguing, and suggests there may be distinct advantages to spending time in a lower-glucose, higher-ketone state of mind.

When insulin is low and ketones are high, your brain shifts into a different metabolic mode—a healing mode that allows it to quiet down and recover from the intensive, insulin-driven operations such as growth, building, and storage that occur when we're actively processing food.

The Yin and Yang of Healthy Brain Metabolism

Scientists at the National Institute on Aging and Johns Hopkins University refer to the shift from carbohydrate-based metabolism to fat-based metabolism as the glucose-to-ketone switch or the *G-to-K switch*. They have documented numerous brain benefits to spending time in ketosis, whether that state is achieved through vigorous exercise, caloric restriction, intermittent fasting, or a ketogenic diet.¹⁵

When you flip your switch from glucose to ketones, a whole host of pathways that have been suppressed by high insulin conditions come to life, including *autophagy* pathways that recycle or destroy damaged cell components (with the help of calcium and zinc), pathways that bolster your immune system and make you more resilient to stress, and pathways that promote *neuroplasticity*, your brain's ability to grow new cells and wire them together in new patterns. Creating these new circuits, which is how we learn and remember new things, requires vitamin A, vitamin D, calcium, and zinc. These duties are essential for optimal brain function, suggesting that *all of us would be wise to spend some time in ketosis on a regular basis*—regardless of what we choose to eat, how metabolically healthy we are, or whether or not we currently have a mental health concern. Keeping your metabolism on its toes by regularly switching up its fuel source appears to be one of the secrets of robust mental and physical health. Unfortunately, many of us have lost our metabolic flexibility. When we look closely at

specific mental and physical health conditions, it will become clear why some of us need to spend most or all of our time in ketosis in order to feel and function well.

DEEPER DIVE: HOW THE BRAIN MAKES ENERGY

To better understand healthy brain metabolism, the kinds of things that can go wrong, and how being in ketosis can help you work around some of those problems, we need to go deeper into the brain cells themselves, where energy is made. This section gets technical, but don't worry about the names of molecules or pathways; they are just there for those of you who want that degree of specificity. Some of the essential micronutrients involved in key pathways are pointed out along the way. If you just want the basics, read the “takeaways” at the end of each concept below.

Concept #1: *Electrons Are Energy*

For a cell to extract energy from any molecule, it must first break open the powerful bonds between the protons (positively charged particles) and electrons (negatively charged particles) that hold their individual atoms together, because that's where the energy is stored. The electrons within glucose and ketones are in strong, stable relationships with their protons, so wrenching them free is no easy task. Free electrons are highly unstable and reactive because they are seeking to return to the safety of a strong chemical bond.

Whether a cell is burning glucose or ketone molecules, the goal is the same: sever their bonds, carefully remove their electrons, capture the energy from those electrons, and store it as *ATP* (adenosine triphosphate) molecules—portable packets of instant energy that cells can easily tear open to power their daily activities. A single busy neuron can burn through more than four billion ATP molecules per second!¹⁶ Neurons use the majority of this ATP to power their sodium-potassium pumps, as well as to create, release, and recycle neurotransmitters, but like all cells, neurons must also tend to a multitude of more mundane duties to support building, storage, maintenance, repair, growth, defense, and reproductive operations, and all of these require ATP, too. Without adequate ATP, your brain cells will not

thrive, and if the energy deficit is too great, they will not survive.

Takeaway #1: To turn glucose and ketone molecules into energy, cells break them apart, remove their *electrons* (high-energy particles), and store the captured energy in miniature ready-to-use power packs called *ATP*.

Concept #2: Cells Have Two Engines

Neurons house two systems for generating energy. One is a simple process called *glycolysis*, which unceremoniously chops glucose molecules in half, quickly releasing small amounts of energy. Let's call this system **engine G**. This ancient metabolic pathway has been used by single-celled organisms since the earliest days of life on Earth when oxygen was scarce.¹⁷ The other is a sophisticated process that completely dismantles fragments of glucose or ketone molecules, generating large amounts of energy. This advanced system requires oxygen and takes place only inside dynamic, double-membraned structures bustling around within our cells called *mitochondria*. We'll call this system **engine M**.

Mitochondria were once independent bacteria-like organisms, swimming freely in Earth's primordial soup along with other microscopic beings, including the single-celled life forms that would ultimately evolve into human beings. Then, some 1.5 billion years ago, one of these mitochondria either invaded one of our microscopic ancestors to live inside of it like a parasite, or one of our microscopic ancestors came along and swallowed one of these mitochondria whole.¹⁸ Either way, all of us now contain these remarkable workhorses within our cells, exploiting them for their energy-producing talent. In a win-win arrangement, we house and feed them and they energize us. Their secret power? Embedded within the innermost membranes of mitochondria are countless *electron transport chains*—marvels of biological engineering that thread electrons through a series of five special protein complexes (I, II, III, IV, and V) to harness energy from electrons and churn it into ATP. Engine M is so powerful that it extracts about fifteen times more energy from glucose than engine G can.¹⁹ Our neurons are so dependent on mitochondria for energy that a single neuron may harbor as many as two million of them.²⁰ Without these hives of electrical activity, we wouldn't be able to produce enough energy to support our complex, multicellular selves, because engine G alone simply

isn't powerful enough.

Burning glucose using engine G is akin to tossing newspaper on a campfire to produce a brief flash of light and heat, whereas burning glucose or ketones inside mitochondria using Engine M is akin to placing a heavy log into an efficient wood furnace and enjoying the light and warmth for hours.

Takeaway #2: Cells contain two engines for making ATP: a simple engine that chops glucose in half to make small amounts of ATP (we'll call this **engine G** because it can only process glucose), and a sophisticated engine that uses oxygen to turn glucose, ketones, and other fuel sources into large amounts of ATP. We'll call this **engine M** because it is located inside *mitochondria*, tiny bacteria-like structures living inside our cells. Engine M gathers electrons from glucose and ketones and then threads them through a series of five special protein complexes called the *electron transport chain* to churn them into ATP.

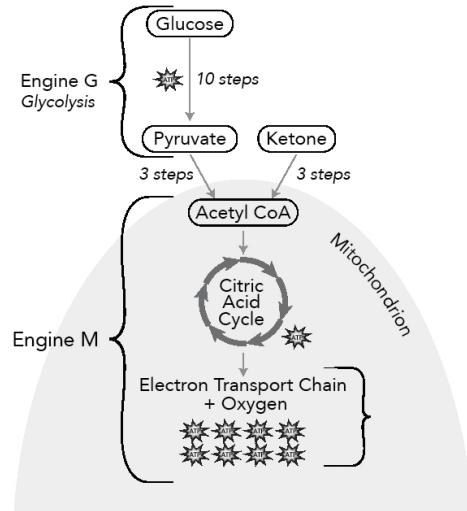
Concept #3: Optimal Glucose Processing Requires Both Engines

To reap the maximum amount of energy from glucose, the neuron must first run glucose through engine G outside the mitochondria, then finish breaking it down inside the mitochondria using engine M. As glucose gets broken into smaller and smaller pieces, more of its electrons are removed:

- Outside the mitochondria, with the help of vitamins B1 and B3, engine G splits glucose in half, creating two molecules of *pyruvate*.
- Pyruvate travels deep into the heart of mitochondria, where vitamins B1, B2, B3, and B5 help break it down into a smaller molecule called *acetyl CoA*.
- Acetyl CoA is the right size to enter the *citric acid cycle*, a rotary of chemical reactions that uses magnesium, iron, and vitamins B1, B2, B3, B5, and B7 to finish completely dismantling it.

Finally, courier molecules gather up stripped electrons from all of the previous steps and escort them to the electron transport chain which uses

vitamin B3, magnesium, iron, sulfur, copper, and oxygen to extract the energy from those electrons and turn it into ATP.



BRAIN ENERGY PRODUCTION SYSTEM

Two systems cooperate to produce the maximum amount of energy. Engine G partially breaks down glucose outside of the mitochondria and Engine M completely breaks down a variety of fuels inside of the mitochondria.

Suzanne Smith

Takeaway #3: Engine M can't burn glucose unless engine G chops it in half first. To extract the *maximum* amount of energy from glucose, cells need both engines: engine G to split glucose into two pieces, and engine M to completely dismantle those pieces.

Concept #4: Glucose and Ketones Burn Differently

Unlike engine G, which can only process glucose, the electron transport chain that drives engine M is an equal opportunity furnace, so it will accept electrons from glucose or ketones.

Just like glucose, ketones must be broken down into acetyl CoA and spun through the citric acid cycle to generate electrons to funnel into the electron transport chain. However, there are key differences between how glucose and ketones are processed that help explain how ketosis helps your brain function better if your brain has trouble burning glucose for energy (which can be the case if you have insulin resistance or certain psychiatric conditions).

1. Ketones burn more quickly and easily because they don't have to go through engine G first.

It takes thirteen chemical reactions to turn glucose into acetyl CoA. Ketones are a more efficient energy source than glucose because it only takes three (completely different) chemical reactions to convert the ketone BHB into acetyl CoA.

Why does this matter?

If any of the steps involved in the long pathway that turns glucose into acetyl CoA is malfunctioning (which may be the case in bipolar disorder, for example; see [chapter 9](#)), you can still make plenty of acetyl CoA out of ketones, because ketone processing completely bypasses all thirteen of those steps.

2. Ketones burn differently because more of their electrons take a shortcut.

As electrons are removed from glucose and ketones, they are handed off to one of two courier molecules to be carried to the electron transport chain: NAD (nicotinamide adenine dinucleotide, made from vitamin B3), which passes its electrons to the first protein complex in the electron transport chain (complex I), and FAD (flavin adenine dinucleotide, made from vitamin B2), which passes its electrons to complex II, a little further down the chain. Engine G uses NAD as its courier more often, which means that engine G traffics more electrons through complex I.²¹

Why does this matter?

If complex I is malfunctioning, which can occur in certain psychiatric conditions such as autism, bipolar disorder, and schizophrenia, then being in ketosis will partially bypass that defect, because electrons that come from ketones don't travel through complex I as often.

3. Burning ketones requires less insulin.

Ketones also come in handy if your brain is short on insulin or if your brain cells have trouble using insulin properly—scenarios that can occur in people with insulin resistance. Under low-insulin conditions, Engine G slows down, but ketones only use Engine M, and ketones burn beautifully in a low-insulin environment.

Takeaway #4: Ketones and glucose burn differently.

1. Ketones burn more quickly and easily because they completely bypass engine G. Also, if engine G is malfunctioning for any reason (which will make it harder to burn glucose for energy), you can still burn ketones using engine M.
2. More of the electrons from ketones take a shortcut into the electron transport chain. Electrons can enter the chain via complex I or complex II. Electrons from ketones use complex II more often, so if there's a problem with complex I, ketones help you partially bypass that problem.
3. Ketone processing requires less insulin. If brain insulin is low or isn't working properly, which can happen if you have insulin resistance, ketones will still burn well; in fact, they burn best under low-insulin conditions.

Concept #5: Two Engines Are Better than One

Ketones have some advantages over glucose, but the brain can't survive on ketones alone. Optimal brain metabolism requires that both engine G and engine M be fully operational.²²

Engine G is useful in situations that require fast energy, in crowded areas inside cells where there is less room for mitochondria, and when blood oxygen supply to the brain has been disrupted (by inflammation, tumor, or stroke, for example).²³

Engine G is also the only entry point for the *pentose phosphate pathway*, which, with the help of vitamins B1 and B2, turns glucose molecules into DNA, RNA, and antioxidants (yes, our cells make their own antioxidants²⁴). In fact, cells can't use glucose for *anything* without running it through engine G first. However, engine G alone isn't powerful enough to generate enough energy to support cell metabolism, and it can't process ketones at all; it can only process glucose.

Engine M efficiently produces large quantities of energy from a wide variety of molecules including glucose and ketones, but it is slower, ill-suited to cramped quarters where there is less room for mitochondria, and performs poorly in low-oxygen environments. Engine M is also vulnerable to damage by the very oxygen molecules it relies on to generate ATP.

Takeaway #5: Engine G and engine M are complementary; each has its advantages and disadvantages, so the cell functions best and produces the maximum amount of energy when both engines are fully operational.

Concept #6: The Oxygen Conundrum

As high-energy electrons from glucose and ketones make their way through the electron transport chain, they gradually give up bits of energy, which complex V turns into ATP. Waiting at the end of the electron transport chain are oxygen molecules which have been cut in half—this allows them to bond with the used electrons and form stable molecules of water. Oxygen's special ability to safely absorb these leftover electrons is the single most important reason why we need to breathe. Without oxygen, electron transport chains grind quickly to a halt, and cells perish.²⁵

Oxygen's love for electrons is a blessing and a curse. When you burn molecules in the presence of oxygen, you're playing with fire, because oxygen molecules love to steal electrons. Whenever rogue electrons prematurely escape the electron transport chain (which they do with some regularity because they are difficult to control), they can react with *whole* oxygen molecules and create highly unstable *oxygen free radicals*. Left to their own devices, free radicals can run amok and damage the cell, a phenomenon called *oxidative stress*. Some degree of oxidative stress is normal and healthy. Mitochondria expect to see a certain amount of oxidative stress and are equipped with their own internal antioxidants to cope with it, so the system has evolved to strike a healthy balance between oxidation and antioxidation. However, if anything interferes with the ability to neutralize free radicals in a timely fashion, or if mitochondria become overwhelmed with too many free radicals at once, the resulting *excessive oxidative stress* can injure mitochondria and jeopardize their ability to produce energy.²⁶ Another important difference between ketones and glucose is that burning ketones results in less oxidative stress.²⁷

Takeaway #6: Engine M uses oxygen, which is risky because oxygen can react with electrons to form unstable substances called *free radicals* that can cause damaging *oxidative stress* to the cell if your cell's own internal antioxidants aren't quickly able to neutralize them. Burning ketones results in less oxidative stress than burning glucose.

In short, optimal mental health requires that both Engine G and Engine M are in good working order. If you feed them the fuel molecules and micronutrients they need, and you protect them from inflammation and oxidative stress, they will serve you well.

NUTRITION FROM THE INSIDE OUT

The point of exploring the microscopic world of human metabolism isn't to memorize the names of pathways and molecules, but rather to marvel at and be humbled by its wisdom and complexity. This infinitely intricate system evolved over millions of years to magically transform whole plant and animal foods into the macronutrients and micronutrients that your brain and the rest of your body need to function at their best, without any help from medications, supplements, nutrition studies, or dietary guidelines.

As complicated as the science of brain metabolism may appear, it reveals some simple truths about macronutrients, micronutrients, and meal timing that must be respected if we are to achieve optimal mental health:

- It bears repeating that the brain requires all essential micronutrients, with those most directly involved in energy production pathways being iron, magnesium, copper, and vitamins B1, B2, B3, B5, and B7. (Thyroid hormone, which contains iodine, is also involved.)
- The brain is a hybrid engine that requires some glucose at all times, but that glucose does not need to come from dietary carbohydrate.
- The brain can't use glucose properly without adequate insulin.
- The brain needs to spend some time in ketosis on a regular basis.

Now that we understand the nutritional and metabolic foundations of robust mental health, the next task before us is to understand the nutritional and metabolic root causes of poor mental health. In other words, what kinds of things can go wrong inside your brain if you don't feed it properly?

PART 2

OUR DESCENT INTO DIETARY MADNESS

CHAPTER 6

The Perils of Processed Foods: Inflammation and Oxidative Stress

Mental power cannot be got from ill-fed brains.

—Herbert Spencer, *Principles of Ethics*

As powerful and sophisticated as your brain's infrastructure is, it is also extraordinarily fragile, and therefore must be carefully protected if you are to enjoy a lifetime of excellent mental health.

Fortunately, Mother Nature has already gone to great lengths to do this for you—she enclosed your brain in a thick layer of bone and triple-wrapped it in membranes to buffer it against physical injury. She even cordoned off its blood supply with a discriminating barrier system to filter out germs and other threatening substances. These ancient evolutionary safeguards lend the brain an air of invincibility, but they are no match for our modern food environment.

What distinguishes today's destructive diets from all those that preceded it? Certainly not the presence of red meat and saturated fat—both of which we've been consuming since time immemorial—but refined carbohydrates and refined vegetable oils. These are the true signature ingredients of the SAD diet, and they have found their way into nearly every processed food on the market. Both of these substances are powerful promoters of excessive oxidative stress and inflammation, two of the driving forces behind the brain diseases we fear most—from depression to dementia and everything in between.

PROCESSING THE WORD “PROCESSED”

Unlike most creatures that simply walk, fly, or swim up to a living plant or animal and swallow it whole, we humans process most plants and animals before we eat them, and have been doing so for millennia. Strictly speaking, anything we do to a plant or animal to fundamentally alter its chemical structure before we consume it—including cooking—is considered “food processing.” So where do we draw the line—is all food processing bad, or is some degree of processing acceptable?

Our love affair with food processing began with the advent of fire hundreds of thousands of years ago,¹ which got our ancestors cooking. After that, nothing much changed on the food preparation scene until some 14,000 years ago—well before the dawn of agriculture—when ancient Jordanians began grinding roots and grains into flour to make bread.² And 7,000 years ago, people on the Adriatic coast of Croatia began curdling milk and straining off the liquid whey through clay sieves to create cheese.³

Processing methods can change foods both for better and for worse. For example, grilling meat destroys some of its vitamins, but also kills potentially harmful bacteria that may be lurking on its surface. Similarly, boiling vegetables destroys some of their vitamins but also reduces their toxicity and makes them easier to digest. Grinding grains improves our access to their nutrients but also to their carbohydrates, leading to greater blood sugar spikes. However, since these time-honored techniques improved the safety, digestibility, or longevity of whole foods, their nutritional advantages largely outweighed their risks. Perhaps this would be a reasonable place to draw the line between “good” and “bad” processing: processing methods with a net benefit to human health? Good. Processing methods with a net risk to human health? Bad. Turning whole foods like beets into pure sugar and converting sunflower seeds into pure oil strips them of their nutritional advantages, leaving us with products that are all risk and no benefit.

What Are Ultraprocessed Foods?

The Industrial Revolution would forever change the nature and purpose of food processing. At first, new machines like rolling mills and new methods like pasteurization simply improved on pre-existing food processing methods, but as we entered the twentieth century, the food industry

gradually moved beyond safety, preservation, and nutritional quality to focus its efforts on *convenience, palatability, and profit*.⁴

We now live in an era of *ultraprocessed* products that would be wholly unrecognizable to our ancestors as food. The Food and Agriculture Organization of the United Nations describes ultraproCESSing this way:

It starts with the fractioning of whole foods into substances including sugars, oils and fats, proteins, starches and fibre.... Some of these substances are then submitted to hydrolysis, or hydrogenation, or other chemical modifications. Subsequent processes involve the assembly of unmodified and modified food substances with little if any whole food using industrial techniques such as extrusion, moulding and pre-frying. Colours, flavours, emulsifiers and other additives are frequently added to make the final product palatable or hyper-palatable.⁵

Ultraprocessed foods make up nearly 40 percent of what Australians choose to consume,⁶ about 50 percent of the food families in the UK purchase,⁷ and more than 60 percent of what Americans buy at the grocery store.⁸ These products are so pervasive that you could probably lay your hands on one right now without leaving your home, car, or workplace—although some of them are easier to recognize than others. Twinkies and Doritos? Clearly ultraproCESSed. But what about CLIF bars and Hidden Valley ranch dressing? These have a natural air about them, but try making them at home and you'll struggle to find enough soy protein isolate or artificial flavors in your kitchen to finish the job.

Most modern processed foods are so nutrient-poor that they can't be considered foods at all, so even the terms "junk food" and "ultraprocessed food" give them more credit than they deserve. Some say products like these are bad for us because they provide nothing more than empty calories—but this is a dangerously inaccurate statement.

Think of it this way: As the storied myth goes, the people of Troy welcomed through their city gates what appeared to be a magnificent hollow wooden horse abandoned by the Greek army during the Trojan War,

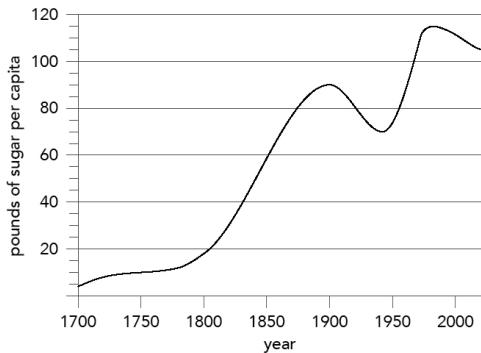
but hiding inside were Greek soldiers that would lay siege to their city. Processed food is the Trojan horse of human health. These products may look innocent and delicious, but concealed within their pleasing packaging are ingredients with the power to destroy your good mental and physical health. Yes, these products are empty in that they lack nutrients—so empty that manufacturers have to *enrich* them with nutrients that their factories have stripped away or *fortify* them with nutrients they never had to begin with. Yet propping them up with vitamins and minerals does nothing to address the fact that they are brimming with toxic molecules that damage your brain’s structure and metabolism over time, ultimately compromising its ability to generate energy.

REFINED CARBOHYDRATES

The term “refined carbohydrates” encompasses a great variety of sweet and starchy ingredients, but the king and queen of this realm, unquestionably, are flour and sugar. The addition of flour to the human menu happened well before written language could record its impact on society, so for a closer look at the power an exciting new source of refined carbohydrates can wield over the human spirit, we’ll consider the more recent tale of sugar.

The Bitter History of Sugar

As Professor Khalil Gibran Muhammad wrote in an article for the *New York Times Magazine*, granulated sugar made its debut about 2,500 years ago in India, but harvesting and boiling sugar cane down into crystals was so labor-intensive that sugar remained a rare delicacy until the 1600s, when Europeans began enslaving human beings from sub-Saharan Africa to toil on sugar plantations in the West Indies, Mexico, Central America, and South America.⁹ Satisfying elite appetites for this “white gold” would ultimately lead to the bondage of millions upon millions of people. This horrific history alone should be enough to convince us all that sugar is not a food, but an addictive drug—much like tobacco, which has a similarly barbaric past.



SUGAR CONSUMPTION IN THE UK

Although it dipped briefly during World War I and World War II, the per capita consumption of sugar (as sucrose) in the UK increased thirteen-fold between 1700–1975.

Data source: Sidney Mintz, *Sweetness and Power: The Place of Sugar in Modern History* (New York: Penguin, 1985), 67, 143, 162, and 198.

This ugly chapter in human history made sugar available to the wealthy Westerners of the 1700s, but as science journalist Gary Taubes writes in *The Case Against Sugar*, it would take the machinery of the Industrial Revolution to convert sugar into an affordable everyday staple for everyone, everywhere: “The industrial revolution, inaugurated by Watt’s steam engine in 1765, transformed sugar production and refining just as it did virtually every other existing industry in the nineteenth century. By the 1920s, sugar refineries were producing as much sugar in a single day—millions of pounds—as would have taken refineries in the 1820s an entire decade.”¹⁰

Defining Refined Carbohydrates

All refined carbohydrates begin life as components of whole plant foods—grains, legumes, fruits, or vegetables—which are then refined into sugars and starches using various industrial processing methods.

Refined sugars are easy to identify. With the exception of honey and fruit juice, any syrup, liquid, crystal, or powder that contains carbohydrate and tastes sweet is refined, including agave syrup, coconut sugar, molasses, and even maple syrup (it takes forty-three gallons of maple sap, which is very low in sugar, to make one gallon of maple syrup).

Most processed foods, including most “savory” foods like soups, salad

dressings, and hot entrees, taste sweet and are full of sugar, even if “sugar” isn’t one of the first ingredients. Manufacturers know that some consumers are trying to avoid products high in sugar, so they have created *dozens* of slightly different forms of sugar and sprinkled them throughout their ingredient lists. In his book *Metabolical*, pediatric endocrinologist Dr. Robert Lustig points out that “by choosing different sugars as the fifth, sixth, seventh, and eighth ingredients, [sugar] can rapidly add up to be the dominant ingredient.”¹¹ How many names for sugar can you find in this ingredient label for a popular name-brand breakfast bar?

Whole wheat flour, invert sugar, whole grain oats, corn syrup, soybean oil, sugar, vegetable glycerin, enriched wheat flour, soluble corn fiber, apple puree concentrate, dextrose, fructose.

The answer is that *seven* of these twelve ingredients are refined sugars—and two of the five remaining ingredients are refined grains, so three-quarters of all ingredients are refined carbohydrates.

SUGAR’S MANY ALIASES

The food processing industry uses more than sixty different names for sugar. Here is a list of the most common types of sugar and some examples of each to help you identify and avoid added sugars in your diet:

- **Sugars** of all kinds (beet sugar, fondant sugar)
- **Juices** (cane juice, fruit juice concentrates)
- **Malt** (barley malt, malt extract)
- **Dextrins** (maltodextrin, tapioca dextrin)
- **Solids** (corn solids)
- **Crystals** (date crystals)
- **Nectars** (agave nectar)
- **Honey**
- **Syrups** (sorghum syrup, brown rice syrup, maple syrup)
- **Molasses**
- **Saccharides** (galactodigasiccharides)

- Ingredients ending in **-ose** (galactose, sucrose)

Also, don't be seduced by flowery descriptions (such as "coconut blossom sugar") or healthy-sounding adjectives—"organic fair trade" sugar is still sugar.

The Whole Truth About Whole Grains

Many of us have become savvy about identifying refined sugars, but refined starches, which come from root vegetables and grains, can be more challenging to recognize. Root starches, like potato starch and cassava flour, are straightforward because they are all thoroughly refined into powders, but refined grains are confusing, because they can be refined *to varying degrees*. White flour is clearly a refined carbohydrate, but what about whole-grain Cheerios, stoneground-rye bread, and brown-rice pasta?

If you've ever been confused about what a whole grain is, you are not alone. Truly whole grains are intact kernels, complete with their outer bran coating, simple as that. Once the grain is broken into pieces by any kind of processing, it is considered refined to some extent. The tinier the resulting particles, the more refined the grain. Particle size matters because the smaller the particles, the easier they are to digest, and the faster your blood sugar will rise after you eat them. Some processing methods produce large, coarse particles (such as cracked wheat and steel-cut oats) that still contain all the grain's original components, whereas other processing methods produce ultra-fine powders (such as wheat flour and corn starch) that have had all of the fiber and most of the nutrients stripped away.

To make matters more confusing, there are some refining methods that don't involve cutting or grinding at all. Examples include polishing (white rice), pressure rolling (rolled oats), and extrusion puffing (puffed wheat). These techniques break down or remove the bran coating of grains, resulting in products that are easier to digest.

But the most insidious reason for widespread confusion about whole grains may be the USDA's definition of whole grains: "Whole grains consist of the entire grain seed, usually called the kernel. The kernel is made of three components—the bran, germ, and endosperm.... For food

products to be labeled ‘whole grain,’ they must contain the same proportions of bran, germ, and endosperm as the original whole grain.”¹²

This absurd policy allows processed food manufacturers to blast a kernel of wheat to smithereens and still call it whole, so long as they add the right ratio of its bits back into the product. To say that this is an unconventional definition of the word “whole” would be an understatement.

Manufacturers are invested in using the term “whole grain” on their product packaging because whole grains are supposed to be healthier than refined grains, and there is plenty of human clinical trial evidence to support this view. Dozens of randomized controlled trials have shown that refined grain products like instant oats and rice pasta lead to higher peaks in blood glucose and insulin levels after meals than traditional oats and brown rice.¹³

The Problem with Refined Sugars and Starches

Your body breaks down the carbohydrates in every food you eat—whether it’s sweet or starchy, whole or refined—into glucose. Since bananas and cotton candy both turn into identical glucose molecules, why does it make a difference which one you eat?

As omnivores, we naturally possess the capacity to eat plant foods like fruits and root vegetables and break their sugars and starches down into glucose—but our physiology never expected to be deluged with large quantities of concentrated carbohydrates multiple times per day throughout our lives. Our pre-historic ancestors may have consumed fruit juice, but it’s unlikely they drank significant amounts of it on a regular basis because most fruits are difficult to juice without special equipment (how long might it take to make even a single cup of apple juice by hand?), and fruits that are easy to juice, like citrus fruits, were historically too sour to be enjoyable.¹⁴ There is evidence that some of our prehistoric ancestors used stones to grind grain, but it’s difficult to imagine them using that crude technique to produce much flour, let alone the 133 pounds of flour per year that the average American consumes.¹⁵

The reason why fresh fruits and vegetables are better for you than candy is that their sugars and starches exist within a fiber matrix, so it takes your body more time to access them and break them down into glucose. They

also contain water, vitamins, and minerals, so they are more nourishing and more satisfying. More than thirty RCTs have found refined carbohydrates to be less filling and less satisfying, and to leave people hungrier than unrefined carbohydrates.^{[16](#)}

EXAMPLES OF FOODS HIGH IN REFINED CARBOHYDRATES

Becoming savvy about refined carbohydrates means reading labels carefully to look for **sugars, flours, syrups, powdered starches, and fruit juice concentrates**.

Products made with grain flours [grain = wheat, corn, rice, oats, barley, rye, buckwheat, amaranth, spelt, millet, etc.]:

- Most breads and baked goods, including gluten-free baked goods
- Most crackers (except those rare brands made entirely from whole seeds and whole grains)
- Most pastas, noodles, dumplings, egg rolls, tortillas, and wraps
- Salad croutons, breadcrumbs, and panko crumbs (most recipes for meatballs, meat loaf, stuffed seafood and stuffed mushrooms use breadcrumbs)
- Batter-fried or breaded foods such as fish sticks, chicken fingers, corn dogs, fried chicken, and eggplant parmesan

Products containing hidden refined starches:

- Most hot and cold breakfast cereals are made from refined grains, including rolled oats, cream of wheat, granola, and bran cereals. Rare exceptions include unsweetened whole grain muesli and cereals made entirely from unsweetened sprouted whole grains.
- Most gravies, sauces, soups, and stews are thickened with

wheat flour or corn starch.

- Refined corn products: grits, corn bread, polenta, nachos, and corn chips
- Refined rice products: rice cakes, rice wrappers, and white rice
- Crunchy snacks made from powdered root starches: vegetable straws, cassava chips, potato sticks, etc.
- Many French fries are now coated with wheat starch, dextrin, or other refined carbohydrates to increase crispiness.

Products containing hidden sugars:

- Many milk substitutes such as soy milk and oat milk are sweetened with sugar.
- Most salad dressings, marinades, barbecue sauces, ketchups, and relishes contain sugar.
- Most smoothies are high in simple sugars like pureed fruit, fruit juice, sugar, or honey.
- Dried fruit snack products such as fruit rolls, fruit gummies, and fruit bars
- Some dried fruits are routinely coated in sugar such as dried cranberries, blueberries, and pineapples.
- All liqueurs and sweet wines contain sugar.

Questionable items (minimally processed, no added sugar, but still high in simple sugars or starches):

- Fruit juices, dried fruit, pureed fruit (e.g. applesauce)
 - Unsweetened milk substitutes made from grains such as oat milk and rice milk
 - Chips made from sliced root vegetables (potato chips, beet chips, plantain chips, carrot chips, etc.)
 - Popcorn, puffed rice, and other puffed grains
-

Refined products like sugars, flours, and juices are essentially concentrated sources of pre-digested carbohydrate that have had their fiber and nutrients stripped away. These naked carbohydrates turn instantly into glucose in your digestive tract and are rapidly absorbed into your circulation, resulting in steeper glucose and insulin spikes in your bloodstream. Remember: Every time your blood sugar spikes, your brain sugar spikes right along with it—and therein lies the problem.

High blood glucose (*hyperglycemia*) is toxic to cells. This *glucotoxicity* explains why people with type 2 diabetes—a disease of persistently high blood glucose levels—risk serious damage to every part of their body from the blood vessels in their eyes to the nerves in their feet. Hyperglycemia has the power to destroy every organ you possess, and the brain is no exception.¹⁷

Concentrating, remembering, and processing information depends on lightning-fast electrical signaling between neurons. When neurons are awash in glucose, they transmit signals more slowly, which may help to explain why people with high blood-sugar levels experience brain fog.¹⁸ But how does excess sugar destroy brain cells?

OXIDATIVE STRESS AND INFLAMMATION: THE BRAIN UNDER ATTACK

High glucose levels jeopardize brain health by promoting excessive oxidation and inflammation.¹⁹ Surplus sugar gloms onto the proteins, fats, and even the DNA strands inside your cells, disfiguring and crippling them beyond repair. These sticky, disabled molecules are called *advanced glycation end products*, or AGEs—an appropriate term, since they are notorious for accelerating the aging process. If you've ever had a hemoglobin A1C blood test to look for signs of type 2 diabetes, then you've tested for AGEs. This test measures how much sugar has become stuck to the protein inside your red blood cells—the more sugar there is in your blood, the more it will attach to everything in your body, including your blood proteins.

Vigilantly patrolling your brain are surveillance cells called microglia that roam their local precincts looking for disturbances, including AGEs. When microglia detect the presence of these caramelized clusters, they

initiate a chain reaction of strategies to destroy them. They begin by unleashing a burst of free radicals (highly reactive oxygen compounds). Like bulls in a china shop, these reckless radicals randomly damage everything they encounter (oxidative stress), setting off local alarms and triggering the release of proteins called *inflammatory cytokines*²⁰ that act as S.O.S signals that set to work to deliberately create inflammation.

Cytokines cross into the bloodstream to alert the rest of the body that the brain is under attack and instruct your whole body to temporarily adopt a new set of priorities to deal with the emergency. Their instructions will depend on the nature of the problem, so, for example, if the problem is sticky AGEs, cytokines might travel to your bone marrow to recruit white blood cells to the scene to help assist microglia in mopping them up. If the problem is a bacterial infection, cytokines might mount a fever to kill off bacteria or initiate a coordinated *sickness response* to make you feel tired, lose interest in activities, and stop eating.²¹ This fasting response helps your body enter healing mode; unless you are malnourished to begin with, eating would just divert precious energy and resources away from the emergency and delay the healing process. Regardless of the threat, once the situation is under control, healing forces will be mobilized to repair any local damage and restore the status quo.

As you can see, your brain's ability to cope with AGEs and prevent them from gumming up the works depends on the deliberate creation of *temporary, controlled* inflammation and oxidative stress. Unfortunately, most people eat or drink refined carbohydrates with every meal and snack, generating dangerous glucose spikes inside the brain multiple times per day and even well into the evening, so AGE production rarely lets up long enough to allow healing to take place. Repeated bouts of free radical exposure can drain your brain of precious antioxidants and ignite a vicious cycle of *chronic, uncontrolled* inflammation and oxidative stress.

It is now well established that chronic brain inflammation plays a significant role in many psychiatric disorders.²² The scientific evidence tying inflammation to depression is especially robust.²³

- Levels of inflammatory cytokines tend to be higher in people with mood and psychotic disorders.²⁴

- Researchers can even “give” people depression by giving them medicines that raise their inflammatory cytokine levels.²⁵
- Inflammatory cytokines disrupt normal production of serotonin and glutamate²⁶—key neurotransmitters involved in mood and psychotic disorders.
- People with depression can feel tired, stop caring about things they usually enjoy, and lose their appetite, just as people do when experiencing a sickness response.
- Adding anti-inflammatory medications to antidepressant medications helps some people with depression feel better.²⁷

Oxidative stress also plays a role in depression, anxiety, bipolar disorder, psychosis, and neurodegenerative conditions such as Alzheimer’s disease.²⁸ Free radicals are dangerous no matter where they are unleashed, but if the neighborhood under attack happens to be your brain, the damage can be particularly severe—the brain doesn’t produce as many antioxidant molecules as other organs do, so its capacity to extinguish free radicals is limited.²⁹ To make matters worse, brain cell membranes are rich in DHA and arachidonic acid—polyunsaturated fats (PUFAs) with fragile double bonds that render them exquisitely vulnerable to oxidative damage.

REFINED VEGETABLE OILS

Excess glucose isn’t the only instigator of damaging inflammation and oxidative stress in the brain we need to be mindful of; other aspects of lifestyle including smoking, vaping, and drinking alcohol are common culprits as well, but most of us recognize these as harmful habits so we wouldn’t expect them to have brain benefits. Unfortunately, this is not the case with vegetable oil. We have long been told that vegetable oils such as soybean and sunflower oil are good for us because they are rich in essential plant PUFAs and free of saturated fat and cholesterol, but these oils are now coming under increasing scrutiny for their potential role in the decline of our mental and physical health.

The Slick History of Vegetable Oil

Unlike sugar, which could be extracted from cane by human hands with hard labor and hot water, most vegetable oils couldn't have existed before the Industrial Revolution, because extracting oil from corn, grape seeds, and other seed crops and refining it into a clear, odorless substance is virtually impossible without heavy machinery and chemical engineers.

As Nina Teicholz chronicles in *The Big Fat Surprise*, prior to the twentieth century, with the exception of olive oil (which was not widely available), oils were not viewed as food: “Oils weren’t even considered edible. They didn’t belong in the kitchen. They were used to make soaps, candles, waxes, cosmetics, varnishes, linoleum, resins, lubricants, and fuels.”³⁰

The first vegetable oil to be slipped into our food supply was cottonseed oil. Extracted from the agricultural waste of the cotton industry, cottonseed oil became a cheap replacement for liquid animal fats like whale oil (used in lamps and candles) and solid animal fats like lard and tallow (used in cooking).

In 1908, as Teicholz goes on to describe, the Proctor & Gamble company patented an industrial method for partially hydrogenating cottonseed oil, which magically transformed it from a toxic, unappealing liquid into a familiar-looking solid. “The original idea at the company had been to employ this new substance to make soap, but the white or yellowish creamy product, which looked so much like lard, also suggested a food use... Finally, [Proctor & Gamble] settled on the name Crisco, derived from its chief ingredient, *crystallized cottonseed oil*.³¹ Clever marketing convinced consumers to welcome Crisco into their kitchens as a clean, modern, and affordable improvement over old-world animal fats.

It wouldn’t be long before corn oil was hardened into margarine as a Depression-era replacement for butter (ultimately inspiring the trendy, plant-based buttery spreads of today). But liquid vegetable oils wouldn’t find their way into our cupboards until the 1960s, after they were endorsed as “heart healthy” by the American Heart Association—a once tiny organization that amassed scientific and political power largely fueled by funding from Proctor & Gamble.³²

Industrially refined vegetable oils like canola (from rapeseed), safflower,

soybean, sunflower, corn, and grape seed oils have now infiltrated nearly every processed food product on the market from salad dressings to potato chips to oat milk.

We've been conditioned to divide fats into saturated and unsaturated categories for so long that learning to divide them into refined and unrefined categories may take a little practice. The fats that exist naturally in any whole plant or animal food are unrefined; examples include a fatty pork chop, an olive, or a macadamia nut. The traditional processing methods used to extract these natural fats, such as cooking pork to render lard or cold pressing olives to make extra virgin olive oil, don't change their chemical structure considerably, so rendered animal fats and cold-pressed oils are not generally considered refined.

You can't, however, wrangle oil out of soybeans or sunflower seeds simply by cooking them on the stove or squeezing them with all your might. The only way to get them to part with their oils is by transporting them to an industrial oil refinery where they will be subjected to a multi-step process requiring hexane—an explosive, petroleum-based solvent. The purpose of all this is "to create an odorless oil with a bland taste and increased storage stability."³³

HOW TO MAKE SOYBEAN OIL IN THIRTEEN EASY STEPS (DON'T TRY THIS AT HOME)

Remove seed hulls, flatten beans into flakes, then pass through an industrial feedscrew extruder to compress the flakes and destroy their cellular integrity.

(Before proceeding to the next step, decrease the atmospheric pressure in the room to reduce the risk that flammable gases will escape and explode.) Next, add hexane, and let the mixture percolate. Place mixture in a desolventizer and heat to 212 degrees Fahrenheit until most of the hexane has evaporated, then let cool. Air dry, then pour into a steam stripper to further concentrate the oil. To prevent unsavory deposits from forming during shipping, degum your hot oil with

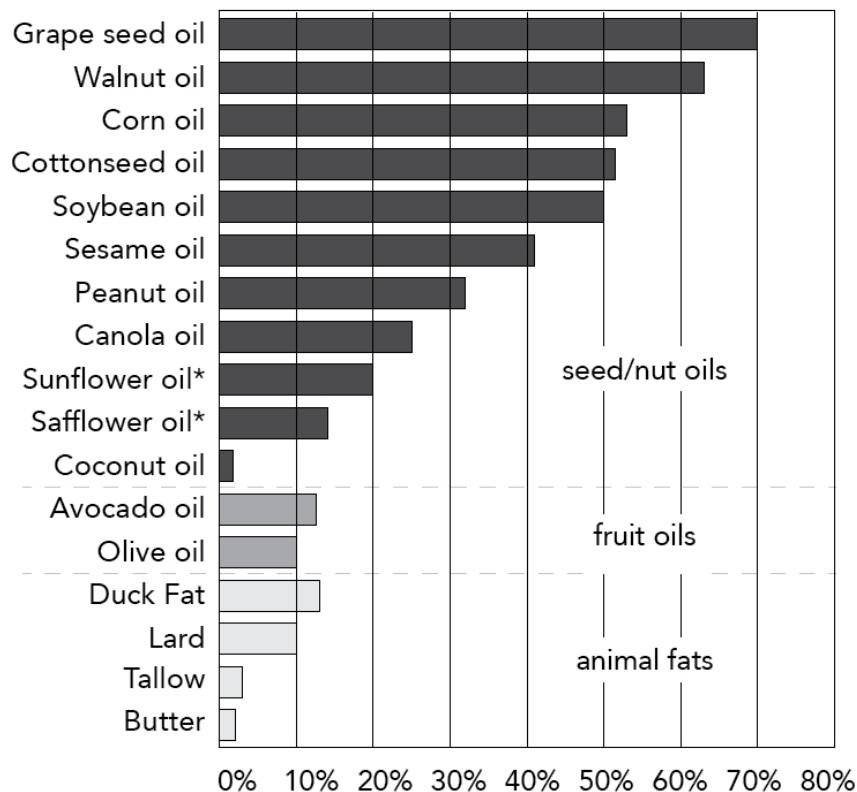
water and set the extracted gums aside. (Important! Do not discard, as these gums can later be sold to your fellow processed food manufacturers as soy lecithin.)

To neutralize your oil, mix with sodium hydroxide (lye) and place in a centrifuge to remove the soap that naturally forms as a by-product. Add a natural bleaching agent such as clay, then filter to remove any soapy residues, pigments, waxes, and rancid fatty acids. To complete the refining process, place in a vacuum deodorizer and heat to at least 356 degrees Fahrenheit. Let cool, pour into bottles, and label “heart-healthy, plant-based vegetable oil.”

The Linoleic Acid Problem

We have been sold vegetable oils because they are cheap, profitable alternatives to animal fats, not because they are good for us. One of the biggest problems with vegetable oils is that most of them are exceedingly high in linoleic acid.

The table below lists the linoleic acid content of common fats; notice that most vegetable oils (oils that come from seeds, nuts, and legumes) are relatively high in linoleic acid whereas most fruit oils (oils that come from the fleshy parts of plants such as avocado oil and olive oil) are relatively low in linoleic acid.



LINOLEIC ACID CONTENT OF FAMILIAR FATS

Sunflower and safflower oil are now available in both high-linoleic and low-linoleic (“high oleic”) formulas. The high-oleic variety has become more common, so that is the form listed in this table.

U.S. Department of Agriculture, Agricultural Research Service, FoodData Central, 2019, <https://fdc.nal.usda.gov>.

Linoleic acid is naturally found in small quantities in a wide variety of whole plant and animal foods, so we did evolve to consume it—but only in small quantities, and only as part of whole foods, not as refined liquids. Prior to the introduction of vegetable oils into our food supply, linoleic acid made up just 1 to 2 percent of our daily calories, whereas today more than 7 percent of the calories we consume is linoleic acid,³⁴ which means **we consume three to six times more linoleic acid now than we did a century ago.**

The result of having consumed vegetable oils in mayonnaise, salad dressings, chips, fries, margarines, and countless other ultraprocessed foods for decades is that our fat cells—which are designed to store saturated fat, not polyunsaturated fat—are slowly filling up with linoleic acid. A 1968

study of members of a hunter-gatherer society living in the Pacific island nation of Tokelau found that their body fat contained less than 4 percent linoleic acid (and the same was true for the pigs and chickens they raised for food). By contrast, in 1959, the percentage of linoleic acid in the body fat of people living in the United States was about 9 percent, and by 2008 had risen to more than 21 percent. (Among Europeans, the level was about 11 percent in 2015.)

The science in this area is still emerging, but there is growing concern among some clinicians and researchers that lining our fat coffers with linoleic acid could potentially lead to increased oxidative stress and inflammation everywhere, including inside the brain.

Linoleic Acid and Excess Inflammation

If your diet is too high in linoleic acid, you may have more trouble making the omega-3 fatty acids EPA and DHA, which could make you more prone to chronic inflammation (DHA helps resolve inflammation in the brain, and EPA helps resolve inflammation in the rest of the body).³⁵ You may not need to worry about this if your diet contains adequate amounts of EPA and DHA, but unfortunately, not only have we been consuming far more linoleic acid than ever before, but we have also been consuming far less EPA and DHA.

DHA and EPA are much harder to find in modern diets than linoleic acid because they don't exist in plant foods at all, and many of the animal foods that contain them in significant amounts (such as shellfish, fatty fish, grass-fed liver, and pastured egg yolk) have fallen out of favor. It is estimated that prior to the dawn of agriculture, our diets contained a much better balance between omega-3 and omega-6 fatty acids—roughly equal amounts of each, or perhaps no more than twice as much omega-6 as omega-3—whereas today, most of us consume at least **twenty times more** omega-6 than omega-3, shifting our entire system toward inflammation and away from healing.³⁶

Mental health researchers have conducted numerous studies trying to correct this imbalance by increasing the amount of omega-3 in the diet (usually with fish oil supplements), often with disappointing results.³⁷ It is surprisingly rare for scientists to approach the imbalance by reducing the

amount of omega-6, but researchers at the National Institutes of Health found that while increasing the amount of omega-3 in the diet (to 1.5 grams per day) for sixteen weeks did improve migraine symptoms, if they also reduced the amount of linoleic acid in the diet (from 7 percent to less than 1.8 percent of calories), migraine symptoms improved even more.³⁸

Linoleic Acid and Oxidative Stress

Linoleic acid is easily damaged by oxidative stress and has a tendency to decompose into toxic byproducts known as *OXLAMs* (oxidized linoleic acid metabolites). There is some research suggesting that OXLAMs may play a role in physical health problems such as heart disease, fatty liver disease, and obesity,³⁹ and although OXLAMs form in the brain as well, they don't appear to accumulate in the brain to any significant extent.⁴⁰ However, this doesn't necessarily mean that high linoleic acid diets are safe for our mental health.

The brain absorbs linoleic acid just as easily as it absorbs any other PUFA, but handles it very differently.⁴¹ Instead of tucking it all neatly into brain cell membranes (as it does with other PUFAs) or turning it into arachidonic acid (which is why we are told we need to consume it in the first place), the brain recycles some incoming linoleic acid into other molecules, but it burns most of it for energy. (This takes place in glial cells because most neurons can't burn fatty acids.) We can only speculate as to why the brain would choose to burn linoleic acid for energy—but it certainly is puzzling, and potentially quite concerning. The brain generally prefers to burn other fuels such as glucose and ketones over fatty acids because burning fatty acids generates many more oxygen radicals, which increases oxidative stress inside the brain.⁴²

In a thoughtfully designed study published in 2022, scientists at Wake Forest University observed that people with Alzheimer's disease had linoleic acid blood levels 56 percent higher than people without cognitive impairment, and the higher the levels were, the less energy their white blood cells were able to produce.⁴³ These researchers speculated that if excess linoleic acid also hampers the ability of brain cells to produce energy, it could directly contribute to the development of dementia and other mental health problems, but they haven't tested this hypothesis yet.

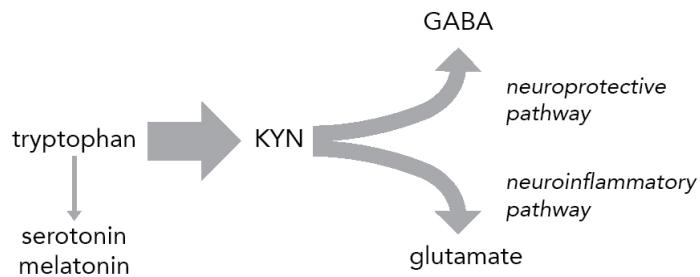
We still have a lot to learn about linoleic acid, but the information we already have available should give us pause. If you share this concern, you have nothing to lose (and potentially a great deal to gain) by removing these industrially refined oils from your diet. In fact, even though linoleic acid has long been considered an essential PUFA, this view is no longer supported by the science.⁴⁴ So long as your diet contains adequate arachidonic acid (which comes from animal foods), you don't need to consume any linoleic acid at all.

INFLAMMATION, OXIDATIVE STRESS, AND NEUROTRANSMITTER IMBALANCES

The increased inflammation and oxidative stress caused by refined carbohydrates and vegetable oils can also contribute to psychiatric problems by throwing your neurotransmitters out of balance—the very same neurotransmitters that most psychiatric medicines are designed to target. Let's take a closer look at how inflammation and oxidative stress change the way your brain uses *tryptophan*, an essential amino acid that comes from dietary protein.

Under normal circumstances, your brain uses some of the tryptophan it absorbs to make *serotonin*, a neurotransmitter which helps regulate mood, sleep, and appetite (among many other things), and *melatonin*, a hormone that helps regulate sleep. The rest of the tryptophan gets directed to the *kynurenine pathway*, where it is used to regulate the production of other neurotransmitters, including glutamate and GABA.

Glutamate is the brain's primary stimulating neurotransmitter and GABA is the brain's primary relaxing neurotransmitter; you can think of glutamate as the brain's gas pedal and GABA as the brain's brake pedal. Glutamate and GABA are the most abundant and widespread neurotransmitters in the brain, and the balance between the two essentially determines your brain's overall activity level at any given point in time. When your tryptophan system is in healthy balance, you will feel calm without being sleepy, and focused without being obsessive.

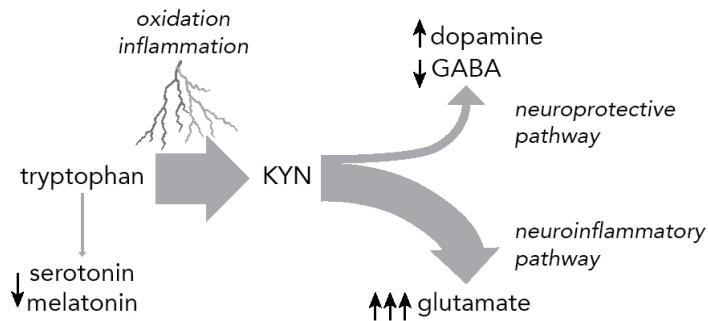


KYNURENINE PATHWAY—BALANCED

The kynureneine pathway under normal conditions.

Excessive inflammation and oxidative stress disrupt this peaceful state and shift your brain into crisis mode by stealing some tryptophan away from the serotonin/melatonin pathway and directing it down the lower branch of the kynureneine pathway. The result of this shift is less serotonin, less melatonin, more dopamine, less GABA, and up to one hundred times more glutamate.⁴⁵ If inflammation and oxidative stress persist, they will lead to *glutamate excitotoxicity*—dangerously high glutamate levels that attack proteins, membranes, DNA, and mitochondria throughout the brain, resulting in widespread structural damage.

Glutamate imbalances are a feature of many psychiatric disorders, including depression, bipolar disorder, schizophrenia, obsessive-compulsive disorder, and Alzheimer’s disease.⁴⁶



KYNURENINE PATHWAY—UNBALANCED

The kynureneine pathway under stress throws neurotransmitters out of balance.

What's Standing in Your Way?

The single most important change you can make to your diet to protect your

brain from damaging inflammation and oxidative stress is to avoid refined carbohydrates and vegetable oils, which starts with eliminating ultraprocessed foods.

It's easy to see why we should stop eating these things, but hard to imagine life without them. Most of us grew up on these intensely flavored, convenient products, and they're designed by chemical engineers to be biologically irresistible. In his book *The End of Overeating*, former FDA commissioner Dr. David Kessler interviewed a leading food industry consultant who told him that the food industry intentionally designs products to hit "the three points of the compass. Sugar, fat, and salt make a food compelling... they make it indulgent. They make it high in hedonic value, which gives us pleasure.... We try to bring as much of that into the equation as possible."⁴⁷

Researchers know that ultraprocessed foods are addictive, and marketing executives shamelessly pitch them to us as exactly that, as if this property is a plus, with slogans like "Bet you can't eat just one" and "Once you pop you can't stop." Food should nourish and satisfy you, not leave you feeling empty, desperate, and out of control; these emotions are the hallmarks of an unhealthy relationship!

Tips for Reducing Inflammation and Oxidative Stress

- **Read ingredient labels.** It's easy to recognize chips and ice cream as junk food, but it can be harder to spot junk foods when they are dressed up as health foods, because they are so often marketed to us based on what they do *not* contain: fat-free salad dressings, sugar-free puddings, dairy-free yogurts, gluten-free cookies, cholesterol-free burgers, etc. It's not enough to know what's *not* in your food; you need to know what's *in* it. Keep your eye upon the donut, not upon the hole. There's no substitute for turning the package over and reading the ingredient label.
- **Cook at home.** Most restaurants, from fast-food joints to fine dining establishments (often proudly) use vegetable oils, so home cooking is always best, but you'll need to clean out your pantry first. If you're like most people, you probably have sugar, flour, and vegetable oil in

your pantry.

Nutrition policymakers around the world continue to recommend that we consume 2 to 10 percent of our daily calories as linoleic acid.⁴⁸ This advice is illogical because linoleic acid is not an essential fatty acid, but even if it were: Unless you have been stranded on a deserted island without access to modern foods for many years, chances are excellent that you have already accumulated enough linoleic acid to last you a good long while. If you were somehow able to eliminate all linoleic acid from your diet, it would take about 680 days to use up just *half* of what you've stored.⁴⁹ but don't let that discourage you—the sooner you begin, the sooner you will get there. Remember, (appropriately) small amounts of linoleic acid exist naturally in a wide variety of whole plant and animal foods, so the chances of your developing a linoleic acid deficiency are vanishingly small.

In the meantime, take comfort in the fact that our capacity to store excess carbohydrate is so small that most people can normalize and stabilize their blood sugar levels within a matter of days by switching to a low-carbohydrate diet—and if you do, chances are excellent that you will be rewarded with less inflammation. In 2022, researchers at the University of Sydney reviewed sixty-three low-carbohydrate clinical trials in humans and found that levels of inflammatory molecules in the blood went down in forty-four of these studies—a robust success rate of 71 percent.⁵⁰ If low-carbohydrate diets aren't your cup of tea, take heart. You can make just about any dietary pattern less inflammatory by avoiding refined carbohydrates.⁵¹

Practicing whole food principles may seem like a daunting task, but your good mental health is well worth the effort. In [chapter 16](#), I'll give you tools and resources to help you practice eating in a whole new way.

CHAPTER 7

Metabolic Mayhem: The Invisible Hormonal Roller Coaster

Perhaps we can recognize our way out of patterns rather than repeating our way out of them.

—Patti Digh, *Life Is a Verb*

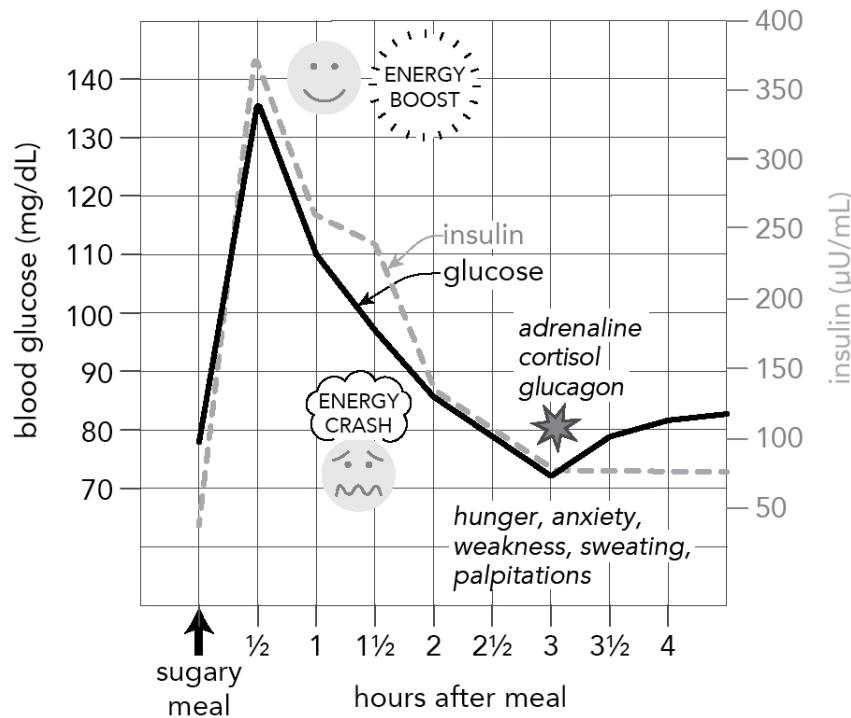
As damaging as high glucose levels are for your brain, glucose is only one half of the story. The other half: Every glucose wave is automatically followed by an insulin wave. If you're metabolically healthy *and* you eat a whole-foods diet, your glucose and insulin levels will rise and fall appropriately. But if you're metabolically healthy and eat too many of the wrong carbohydrates too often, or if you're metabolically unhealthy and eat more carbohydrate (of any kind) than your system can handle, your glucose and insulin patterns will be abnormal. Depending on how sugary your diet is and how damaged your metabolism is, you could see anything from glucose and insulin levels poking just above their usual safe limit to dramatic spikes and crashes that destabilize you from within.

Remember that insulin is more than just a blood glucose manager; it's a master growth hormone, which means that it influences many other hormones, including hormones that regulate appetite, stress, reproductive cycles, and even blood pressure. Every time insulin rises and falls, it takes all of those other hormones along for the ride. Since one of insulin's responsibilities is to squirrel away incoming glucose molecules into your cells, the more carbohydrate you eat, the more insulin it will take to bring your glucose back down to normal, so the *higher your blood glucose, the higher your blood insulin*. Unnaturally steep insulin spikes can mean unnaturally steep drops in glucose, which can trigger complicated hormonal

reactions.

SUGAR AS A MOOD DESTABILIZER

If your glucose is falling too fast or from too great a height, your glucose-hungry brain will wisely view your plummeting glucose as an emergency and respond by commanding your body to release a cocktail of hormones into your bloodstream to keep your glucose from dropping to dangerously low levels.¹ This mixture includes appetite hormones that urge you to eat, as well as glucagon and adrenaline, which tell your liver to release more glucose into the bloodstream.



HORMONAL ROLLER COASTER

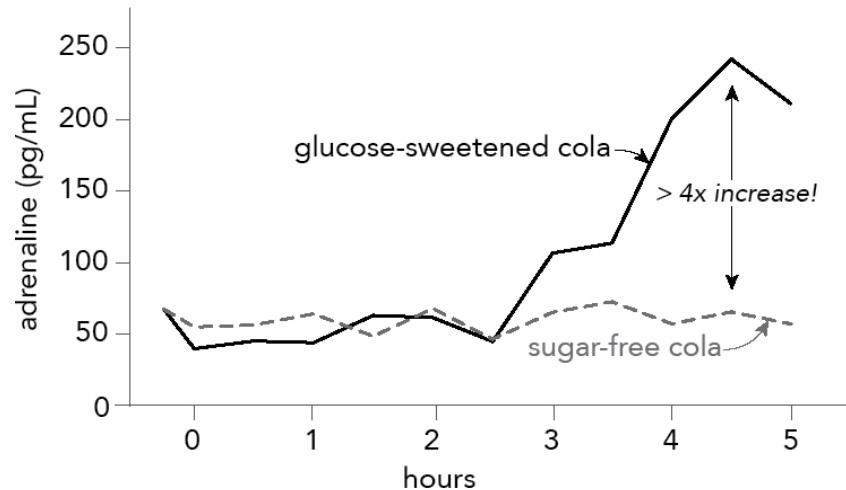
Glucose and insulin spikes trigger stress hormone reactions.

Data source: M. E. Daly et al., "Acute Effects on Insulin Sensitivity and Diurnal Metabolic Profiles of a High-Sucrose Compared with a High-Starch Diet." The American Journal of Clinical Nutrition 67, no. 6 (1998): 1186–96,
<https://doi.org/10.1093/ajcn/67.6.1186>.

Glucagon and adrenaline take care of the falling glucose issue, but adrenaline does much more than raise blood glucose—it is a powerful stress hormone that prepares you to cope with physically challenging or perilous

situations. Adrenaline injects glucose and fat into your bloodstream for energy, shifts blood flow away from your digestive system and toward your large muscle groups, and stimulates your heart to pump more vigorously, all to prime you for “fight or flight.” This hormonal reflex can be useful if you’re in physical danger, but if all you’ve done is enjoyed a couple of cans of soda while watching TV, you could feel as if you’re having a panic attack out of the blue for no good reason.

In the experiment below, researchers at Yale University gave healthy teenage boys a decaffeinated cola sweetened with 86 grams of glucose—about the same amount of sugar you’d find in two twelve-ounce cans of soda. Four to five hours after the boys drank the sugary cola, their adrenaline levels *quadrupled*, and they reported symptoms such as shakiness, sweating, weakness, and pounding heart.²



EFFECT OF SUGAR ON HEALTHY BOYS

Glucose-sweetened cola triggers dramatic surge in adrenaline levels.

T. W. Jones et al., “Enhanced Adrenomedullary Response and Increased Susceptibility to Neuroglycopenia: Mechanisms Underlying the Adverse Effects of Sugar Ingestion in Healthy Children,” *The Journal of Pediatrics* 126, no. 2 (1995): 171–7, [https://doi.org/10.1016/s0022-3476\(95\)70541-4](https://doi.org/10.1016/s0022-3476(95)70541-4).

Since it takes nearly five hours for these sugar-driven adrenaline waves to wash over you, it’s not always easy to make the connection between how you feel and what you ate and drank so much earlier. This emotional and physical reaction to going without food for five hours has become such a common phenomenon that it’s earned itself the clever nickname “hangry”

(hungry plus angry), but it is not normal. If you sometimes feel uncomfortably hungry, anxious, or unstable between meals, the simplest thing to try first is to remove the refined carbohydrates from your diet.

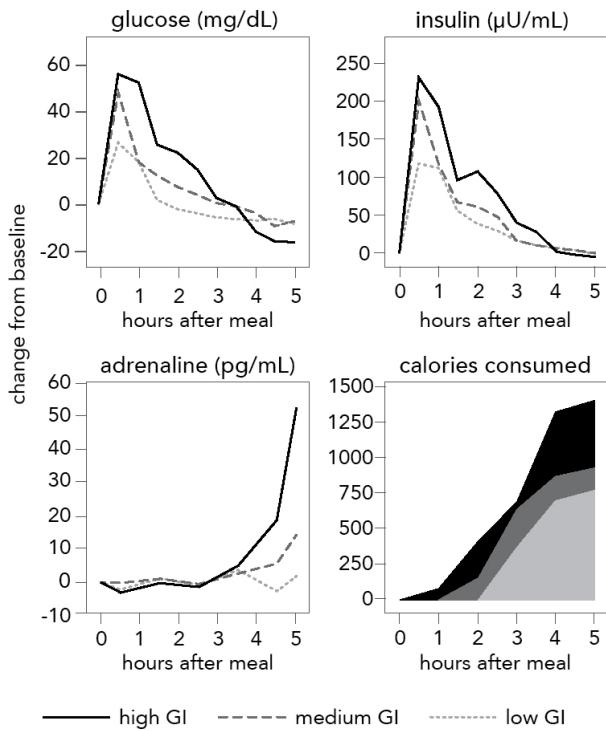
Harvard endocrinologist and obesity prevention scientist Dr. David Ludwig showed that removing sugar and refined grains was all it took to prevent dramatic spikes in the adrenaline levels of overweight teenage boys.³ His research team tested three breakfasts, each one with a different *glycemic index*, or GI for short (the glycemic index is an estimate of how strongly foods raise blood glucose):

High-GI breakfast: instant oatmeal with table sugar (glucose + fructose) and milk

Medium-GI breakfast: steel-cut oatmeal with fructose and milk

Low-GI breakfast: vegetable-cheese omelet and fresh fruit

The sugary instant oatmeal breakfast triggered a dramatic rise in adrenaline—*much* higher than the steel-cut oatmeal breakfast—even though both meals contained the same amount of carbohydrate. The low-GI breakfast, which contained no grains or sweeteners at all (and was somewhat lower in carbohydrate as well), was gentlest on all three hormones. In his book *Always Hungry*, Dr. Ludwig explains, “Adrenaline surged at four hours after the instant oatmeal... suggesting the brain had experienced a *true metabolic crisis*. Some of our participants looked frankly sweaty and shaky.”⁴ The boys who had eaten instant oatmeal also felt hungrier, and, when given free access to platters of food, consumed far more calories over the next five hours.



HIGH-GLYCEMIC-INDEX BREAKFAST INCREASES GLUCOSE, INSULIN, ADRENALINE, AND APPETITE

D. S. Ludwig *et al.*, "High Glycemic Index Foods, Overeating, and Obesity," Pediatrics 103, no. 3 (1999): E26, <https://doi.org/10.1542/peds.103.3.e26>.

A Food-First Approach to Anxiety

Eating too many of the wrong carbohydrates too often can send insulin soaring, setting off a vicious cycle of hunger, overeating, and mood swings that lead some people to seek help from psychiatrists. Depending on how often these episodes happen and how much distress it causes you, a psychiatrist might diagnose you with panic disorder or binge eating disorder and might recommend cognitive-behavioral therapy or prescribe anxiety medications. But why not look at food first?

A thirty-one-year-old Mexican-American post-doctoral student came to me requesting help with frequent panic attacks, irritability, constant food cravings, "emotional eating," and sleepiness occurring two hours after meals. She was very health-conscious and hoped to avoid medication, so I recommended she try a whole-foods, low-carbohydrate approach. She changed her diet from this:

Breakfast: toast with peanut butter or Nutella, coffee with skim milk
Lunch: salad with tuna or cheese and a piece of bread
Dinner: pasta with cheese
Favorite snacks: bananas and yogurt

to this:

Breakfast: two eggs with butter and guacamole
Lunch: meat and non-starchy vegetable
Dinner: meat and non-starchy vegetable
Snacks: nuts and cheese

When asked how the new diet had affected her symptoms, she told me: “I don’t know how I dealt with it because they all used to bother me A LOT, but I would say that the symptoms are 90 percent gone.” Her original diet was simpler and lower in ultraprocessed foods and sweets than most I’ve seen in my career, yet it still contained too much refined carbohydrate (bread, chocolate, and pasta) to be safe for her metabolism.

Notice she used the term “emotional eating”—a common issue that can be very upsetting to so many people who feel they cannot control their urge to eat when they are under stress. It feels for all the world as if the stress is what is driving the desire to eat, when in most cases, it is the other way around: eating too many of the wrong carbohydrates too often causes a rise in stress hormones that urge us to eat to stabilize our metabolism.

In addition to adrenaline, falling glucose triggers the release of another stress hormone called *cortisol*. Unlike adrenaline, which is a simple, fast-acting hormone for sudden emergencies, cortisol is a sophisticated, slow-acting hormone that prepares your metabolism, your cardiovascular system, your immune system, and your brain for prolonged periods of stress. Rising cortisol tells your liver to make more glucose at all costs, even if it has to break down your muscle proteins to do it. Cortisol also weakens your immune system, raises your blood pressure, and carries stress messages to your brain that affect neurotransmitter balance and can damage the hippocampus (the brain’s learning and memory center). Unstable or overactive cortisol is a common feature of many psychiatric conditions

including depression, anxiety, insomnia, bipolar disorder, schizophrenia, post-traumatic stress disorder, and dementia.⁵

Understanding Hormonal Havoc

If you're like most people, you're probably including refined carbohydrates with every meal too, and perhaps snacking on them between meals as well, which could mean as many as six insulin spikes per day. Eating this way puts you on a hormonal roller coaster all day long and well into the night that can have a profound impact on your mood, energy, sleep, appetite, and multiple other aspects of your health and well-being.

Other hormones that ride on the glucose-insulin roller coaster include appetite hormones like *ghrelin*, which your stomach releases to make you feel hungry, and *leptin*, which your fat cells release to make you feel full. Female reproductive hormones like *estrogen* also pay close attention to glucose and insulin levels so they can decide whether the conditions are right for pregnancy. When insulin rises, so can *aldosterone*, a hormone that tells your kidneys to hold on to more water and salt. This may cause you to retain fluid or even contribute to high blood pressure, especially if you have insulin resistance or type 2 diabetes.⁶

Unstable glucose and insulin can destabilize all of these hormones, increasing the risk for binge eating, blood pressure fluctuations, susceptibility to infection, and irregular menstrual cycles. Your age, metabolism, gender, body composition, genetics, diet, activity level, medications, and existing health issues all influence what your personal roller coaster looks and feels like, but **most of us eventually pay an emotional or physical price for eating a high-insulin diet.**

We tend to think of our hormones as controlling our eating behaviors—and this is partly true—but as you can see, the whole truth is that our eating behaviors control our hormones, too, so the relationship goes both ways. Fortunately, you have a tremendous degree of control over your insulin levels. While there's no way to monitor your own insulin at home yet, it is easy to monitor your own glucose. Since high glucose is the main driver of high insulin, keeping your glucose in a healthy range is a powerful way to protect yourself from hormonal havoc.

MASTER YOUR METABOLISM USING HOME GLUCOSE MONITORING

If you have mood, concentration, or energy concerns that come and go, and you are curious about whether your food choices may be contributing to them, it is well worth testing your blood glucose at home to look for potentially problematic patterns.

There are two ways to measure your glucose at home—with a fingerstick glucose meter or a continuous glucose monitor (CGM).

Fingerstick glucose testing involves pricking your finger to produce a drop of blood, placing that drop onto a test strip, then inserting it into a handheld meter that tells you what the glucose level of your blood is at that moment. The meters and strips are inexpensive, widely available, and don't require a prescription.

Continuous glucose monitoring measures your glucose around the clock using a painless sensor patch that adheres to the surface of your skin. Each patch lasts for ten to fourteen days. CGMs are available without a prescription in Canada and many European countries, but in the United States, they still require a prescription and aren't usually covered by health insurance. In the United States, patches start at around \$70 each, and if you can afford it, even a single patch will give you up to two weeks' worth of valuable insight into your metabolism. If you can't obtain a CGM prescription from your doctor, there are online direct-to-consumer CGM subscription plans that include prescription services, but these are more expensive.

If you're using the fingerstick method, it is helpful to check your glucose one to three times per day, and the best times to test are about one hour after you eat a meal that includes your favorite carbohydrates to see how your metabolism responds. You should also check your glucose any time you feel shaky, moody, unfocused, or otherwise unwell. Once you have figured out how to eat to keep your glucose in a healthy range, you may not want or need to monitor your glucose very often, if at all, so think of this phase as an up-front investment in your health that will pay off in the long run by saving you money on medications, medical procedures, and clinic visits.

Please note: The suggestions that follow are intended only for non-pregnant adults without type 1 or type 2 diabetes. If you are pregnant, have diabetes, or take any medications for blood sugar problems, please consult with a health care professional for personalized guidance about blood glucose monitoring and target glucose ranges.

Metabolic Red Flags

The three most important things to look for when you're monitoring your glucose are:

1. Glucose highs (hyperglycemia):

Mild to moderate hyperglycemia (prediabetes range) Levels between 140 mg/dl and 199 mg/dl are considered “prediabetes” and are therefore very worrisome. Technically, prediabetes means your blood glucose is abnormally high—just not high enough yet for you to be officially diagnosed with type 2 diabetes. As we’ll discover in the next chapter, even if you never develop type 2 diabetes, prediabetes itself is already a dangerous metabolic disease that deserves your full attention.

Severe hyperglycemia (diabetes range) Levels of 200 mg/dl or higher are dangerous. If your glucose is rising into the diabetes range, contact a health care professional for additional guidance and support.

2. Glucose lows (hypoglycemia):

Mild hypoglycemia Glucose levels between 55 and 69 mg/dl fall into a gray area because some people tolerate them well and others don’t. If your glucose falls into this range and you experience any symptoms of hypoglycemia, such as anxiety, nausea, shaking, sweating, dizziness, headache, hunger, racing heart, confusion, or poor coordination, drink 1 tablespoon of fruit juice, recheck your glucose in 15 minutes, and consult a health professional. If your glucose falls into this range but you feel fine, there may be nothing to worry about; it may simply mean it’s time to eat—but it’s always

a good idea to run it by a medical professional to be sure.

Severe hypoglycemia Your blood glucose should never drop below 55 mg/dl—this is the definition of true, medically concerning hypoglycemia and can be dangerous. Levels this low are most likely to occur in people who take medications that lower blood glucose (such as insulin). If your glucose ever drops below 55 mg/dl, drink 1 tablespoon of fruit juice, recheck your glucose in 15 minutes, and consult a health professional immediately, whether you notice symptoms of hypoglycemia or not (because some people can't sense low blood sugar).

3. **Glucose instability:** Glucose levels that swing up and down frequently or unpredictably are unhealthy and usually mean that you are eating too much carbohydrate for your personal metabolism. Even if your glucose stays within a healthy range all the time, sudden spikes or drops of 30 mg/dl or more can make you feel unstable, tired, or anxious, and puts too much stress on your delicate insulin signaling system, which can lead to insulin resistance or type 2 diabetes over time.
-

HYPOGLYCEMIC SYMPTOMS WITH NORMAL BLOOD GLUCOSE?

Sometimes people experience symptoms that feel like hypoglycemia even though their blood glucose hasn't fallen below 70 mg/dl. In most people, symptoms of "hypoglycemia" don't occur because glucose is *too low*; they occur because glucose is falling *too fast or from too great a height*, causing their stress hormones to overreact. In these cases, the symptoms aren't being caused by low blood sugar; they are caused by stress hormones the body releases to *prevent* low blood sugar. Imagine a frustrated parent who has to repeatedly pull their toddler down off the kitchen counter, so they won't burn themselves on the stovetop. The toddler may experience it as unpleasant and jarring, but it is happening for

the child's own good. This protective hormonal reaction is fairly common and usually means that there are too many refined carbohydrates or too much carbohydrate in general in your diet for your metabolism to safely handle. If this happens to you, have something to eat so you'll feel better, but consider changing your diet to prevent it from happening again.

Achieving Healthy Glucose Goals

Remember, every time your blood glucose spikes and crashes, your brain glucose follows suit. Stable blood glucose means stable brain glucose—and that will go a long way toward improving your peace of mind.

Your morning fasting glucose (before you've had anything to eat or drink except water) should be no higher than 99 mg/dl (between 70 and 85 is ideal). Many factors contribute to high fasting glucose. If your morning fasting glucose is too high, you may have eaten too much carbohydrate the day before or eaten too close to bedtime the night before. If your fasting glucose is consistently above 125 mg/dl consult a health care professional.

Any food that drives your glucose to 125 mg/dl or higher should be avoided. Aim to spend most of your day between about 70 mg/dl and 100 mg/dl. Keep your glucose below 125 mg/dl at all times, including 1–2 hours after meals, when it tends to be at its highest.

Fasting Glucose (mg/dl)	After-Meal (Postprandial) Glucose (mg/dl)
Ideal: 70 to 85	Best ≤110
Normal: 70 to 99	Better <125
Prediabetes range: ≥ 100	Good <140
Diabetes range: >125	Prediabetes range ≥ 140 Diabetes range ≥ 200

If you see unhealthy peaks and valleys in your glucose level, adjust the quality and quantity of carbohydrates in your diet until your glucose quiets

down and remains in a healthy range at all times. You can start by simply removing all the refined carbohydrate from your diet, which all of us should do anyway. If that doesn't help enough, you'll next want to reduce the total amount of carbohydrate in your diet. How low you'll need to go will depend on your metabolism—there's more detailed guidance about dietary changes in [chapter 16](#).

Connecting the Dots

Home glucose monitoring is a powerful tool that can show you whether the mental or physical symptoms you are having are related to your blood glucose levels. For example, my colleague Dr. Penny Figtree used a CGM to identify and solve problems with her glucose levels that had been causing lifelong discomfort. As she describes,

Porridge for breakfast has always seemed a very healthy breakfast option and one I had enjoyed for most of my life. However, one day, whilst wearing a continuous glucose monitor, I saw my blood glucose literally shoot up to almost 12 mmol/L [215 mg/dl] and then drop back down to below 4 mmol/L [72 mg/dl]. This was half an hour after eating some unsweetened porridge. I was shocked! After repeated experiments (which my daughter used for a science project!), I learnt that a 35-gram sachet [2/5 cup] of unsweetened traditional oats (100% Uncle Tobys whole grain rolled oats with water) always caused a spike to over 11 mmol/L [over 200 mg/dl] and then a sudden drop back down. Even steel-cut oats caused a rise to around 10 mmol/L [180 mg/dl]. Plain rice causes this spike too! The wonderful thing about this discovery is I have cured my lifelong reactive hypoglycaemia! These spikes and then drops in blood glucose trigger symptoms of shaking, tremulousness, heart palpitations, brain fog, and tunnel vision.

For my entire life I have dreaded this feeling randomly coming on but now I am cured. All it takes is mostly eating low carb and especially avoiding porridge for breakfast.

—Dr. Penny Figtree, MBBS (hons1) FRACGP, Port Macquarie, Australia

It's not always this straightforward, but in Dr. Figtree's case, all it took to cure decades of perplexing symptoms was a little curiosity, a CGM, and reducing her carbohydrate intake. I'll share more examples of how changing your diet can change your mind throughout the book.



DR. FIGTREE'S CGM READINGS

Dr. Figtree's glucose response to traditional (rolled) oats and steel cut oats.

SUGAR: THE OTHER WHITE POWDER?

It's easy to understand why it's important to normalize glucose and insulin levels, but that doesn't mean it's easy to do. Looming behind the glucose-insulin metabolic roller coaster is the ominous shadow of the roller coaster of addiction: craving, consumption, glucose spike, bliss, insulin spike, glucose drop, craving. Many of us are addicted to sugar and don't realize it. Those of us who have struggled with unmistakably addictive behaviors such as binge eating, intense cravings, and food secrecy acknowledge (at least to ourselves, if not publicly) that we have a problem, but for most people, sugar addiction can be difficult to recognize. This is because we have been taught to shamelessly love this substance from a very young age by well-meaning adults who, more often than not, are themselves addicted to sugar. We enjoy it, celebrate it, and expect to consume it every day. As Gary Taubes writes in *The Case Against Sugar*:

Imagine a drug that can intoxicate us, can infuse us with energy, and can do so when taken by mouth...

Overconsumption of this drug may have long-term side effects, but there are none in the short term—no staggering or dizziness, no slurring of speech, no passing out or drifting away, no heart palpitations or respiratory distress. When it is given to children, its effects may be only more extreme variations on the apparent natural emotional roller coaster of childhood, from the initial intoxication to the tantrums and whining of what may or may not be withdrawal a few hours later.⁷

As both a plant-derived source of carbohydrate-based energy and a purified crystalline substance that reliably delivers instantaneous emotional gratification to the user, sugar blurs the lines between food and drug, making it just as challenging for addiction researchers to convict as for nutrition authorities to defend. The emerging science of sugar addiction tells us that sugar triggers the brain's reward circuits to release *dopamine*, a neurotransmitter that signals pleasure and motivates us to seek more of the same. Repeated use of sugar can eventually dull the dopamine pleasure response, leading to compulsive overeating. Sugar also delays the release of *acetylcholine*, a neurotransmitter that (among other duties) signals satiety in the brain—essentially taking down the stop signs and encouraging nonstop eating.⁸ After reviewing the available research in this field, scientists at the University of Bordeaux concluded that “At the neurobiological level, the neural substrates of sugar and sweet reward appear to be more robust than those of cocaine.”⁹

Because sugar hijacks the hormones and neurotransmitter pathways that regulate your appetite and eating behaviors, when you are eating a diet that includes refined carbohydrates, *you cannot trust your instincts*. This is why the concept of “intuitive eating” fails so many people. Indeed, in a 2022 randomized controlled trial of 58 adults with obesity, an intuitive eating program made no difference in binge eating, emotional eating, or weight.¹⁰

As compelling as the science sounds, most of the research in this field has been conducted in animals, not people, leaving the door open to skepticism. Academics continue to debate whether sugar and other types of food addiction are real, but regardless of where the science may lead, it will

never be able to tell you if you are addicted to sugar; only you can know that.

Removing refined carbohydrates from your diet is key to improving and protecting your metabolic health, and therefore your mental health. If you have any trouble eliminating sugar (and all of the other refined carbohydrates that turn rapidly into sugar in your body), you may be more attached to sugar than you realized. Unawareness of sugar addiction is common, partly because unawareness of sugar consumption is common. Many of my patients earnestly tell me that their diets are low in sugar because they don't eat sweets, drink sugar-sweetened beverages, or add sugar to anything, but refined carbohydrates are so ingrained in our diets that most people eat them multiple times a day without realizing it. Sugar comes in a great many forms, some of which aren't even sweet. Bread, potato chips, pizza, pretzels, and polished grains are all high in refined carbohydrate and can be just as addictive as candy. As one woman I consulted with confided: "I could do a face plant in a bowl of rice."

I encourage you to explore your own relationship with sugar and other addictive ultraprocessed foods. Consider the following questions, taken from the modified Yale Food Addiction Scale (mY-FAS).¹¹ (Find a link to the full questionnaire in appendix A.)

- Do you sometimes have difficulty controlling your intake of certain foods?
- Do you find yourself consuming certain foods even though you are not hungry?
- Do you sometimes feel sluggish or fatigued from overeating?
- Does your eating behavior cause you significant distress?
- Have issues related to food and eating interfered with your ability to function effectively?
- Do you keep consuming the same types or amounts of food despite significant emotional and/or physical problems related to your eating?

If you've identified any hallmarks of addictive eating, you'll find tools to help set you on the road to recovery in part 4.

KNOWLEDGE IS POWER

As tasty, convenient, and fun as it may be to keep eating refined carbohydrates on a regular basis, that way of life comes at a steep price that may include mood disturbances, addictive eating, physical disability, and early death. Understanding how the foods you eat affect your metabolism and mental health empowers you to make healthier decisions that can change the course of your future.

CHAPTER 8

Insulin Resistance: Your Brain's Silent Enemy

Wouldn't it be nice if the human body had an early alert system that advised us when something was about to go wrong with our health? Prediabetes offers a warning and gives us a chance to change the future.

—Diabetes Canada

The road to metabolic ruin is long, slow, and *seemingly* silent. So silent, that most families who experience health crises feel they've been ambushed by a tragedy they never saw coming. Mothers in their forties learning they have breast cancer. Husbands in their fifties dying from heart attacks. Grandparents in their seventies absorbing the dreaded diagnosis of early Alzheimer's disease.

When families share the devastating news about their loved ones, the phrase I hear most often is "But I don't understand why this happened... they were so healthy." They will then list signs of good health: "Mom is a strict vegetarian who walks religiously every day." "My husband biked a hundred miles a week and his cholesterol was perfect." "Grandma is active in her community and does crossword puzzles every morning to keep her brain sharp; she had just had her annual physical and everything was completely normal." Families and medical professionals alike chalk these catastrophes up to genes, age, or bad luck, but what all of these conditions have in common is insulin resistance—a metabolic disease most people have never heard of, most doctors don't test for, and most of us already have.

Insulin resistance has reached epidemic proportions in the United States

and in many other countries around the world. According to the Centers for Disease Control, ninety-six million Americans have insulin resistance, and seventy-eight million of them don't know it.¹ Another thirty-seven million Americans have full-blown type 2 diabetes,² which is a severe, advanced stage of insulin resistance. Taken together, these statistics tell us that 52 percent of Americans have a significant degree of metabolic dysfunction. The United States outpaces most other nations in this regard,³ but the sad truth is that metabolic health is on the decline everywhere in the world. Seventeen percent of Australians,⁴ 19 percent of Canadians,⁵ 25 percent of New Zealanders,⁶ and 27 percent of people living in the UK have insulin resistance,⁷ and these statistics don't even include those with type 2 diabetes.

Insulin resistance develops gradually and has usually already been present for many years before tragedy strikes. Unfortunately, there is no single test for insulin resistance, so we have to diagnose it in roundabout ways, but it's easy to find if you know what to look for. In this chapter, I'll show you how to evaluate yourself for insulin resistance using simple measurements you can do at home and simple blood tests you've probably already had. Once you learn to recognize the telltale signs of insulin resistance, you'll be able to see most medical tragedies—including those of a psychiatric nature—coming from a mile away—and more importantly, you can take action to prevent them.

Phase One: Metabolic Instability

In the early stages of metabolic dysfunction, *instability* is the name of the game: whenever your sugary diet tries to drive your blood glucose up too high, your pancreas releases as much insulin as it takes to pull it back down again. How does insulin accomplish this? It binds to receptors on muscle and fat cells, and those cells respond by pulling glucose out of your circulation. Insulin also binds to receptors in the liver, signaling them to stop releasing glucose into the bloodstream. For a while, these cells respond dutifully to insulin's instructions, keeping your glucose in a healthy range.

Phase Two: High Insulin Levels, Non-Diabetes Glucose Levels

However, if you continue to eat in an unhealthy way, you'll be repeatedly

bombarding your cells with insulin, which puts tremendous pressure on your insulin signaling system. Our cells simply weren't designed to absorb and process large quantities of glucose multiple times per day. To pace themselves and protect themselves from overstimulation, your cells push back and become increasingly *resistant* to insulin's instructions. Over time, a vicious cycle takes hold: the more resistant your cells become, the more insulin it takes to overcome that resistance, and the longer it takes to bring your glucose back down to normal. During this intermediate stage of metabolic malfunction, *high insulin* is the name of the game: instead of quickly going up and then right back down again, insulin goes up higher and stays up longer. As the disease progresses, insulin may need to remain high virtually all the time to cope with incoming glucose loads. This stage of metabolic dysfunction is insulin resistance.⁸

INSULIN RESISTANCE: WHAT'S IN A NAME?

Insulin resistance goes by several names, each with its own strengths and weaknesses.

"Prediabetes" is commonly used because insulin resistance often (but not always) leads to type 2 diabetes.

"Metabolic syndrome" refers to a cluster of familiar health problems that often go hand-in-hand with insulin resistance: increased belly fat, slightly elevated fasting blood glucose, high blood pressure, high triglycerides (blood fats), and low HDL cholesterol (the so-called good cholesterol—see [chapter 11](#) for more information about cholesterol). There is more than one official definition of metabolic syndrome, but the most commonly used diagnostic criteria are listed below. Do any of these apply to you? Even though the official diagnosis of metabolic syndrome requires that you have at least three of these, every one of them is a sign of poor metabolic health, so your goal should be to get as close to zero as you can.

Metabolic syndrome (3 or more of the following):⁹

- Waist circumference: men > 40 inches, women > 35 inches
- Fasting blood glucose between 111–125 mg/dl or taking medication for high blood glucose (higher than 125 mg/dl means you have type 2 diabetes)
- Blood pressure >130/85 (or taking blood pressure medications)
- Fasting triglycerides ≥ 150 mg/dl
- HDL cholesterol: Men <40 mg/dl, women <50 mg/dl

“**Insulin resistance**” is the term I chose to use throughout this book, but it has its shortcomings as well. The first is that it suggests that insulin resistance itself is the primary problem when in reality, insulin resistance begins as a healthy reaction to an unhealthy situation—insulin levels that are too high. (Overexposure to any hormone will naturally result in hormone resistance.) Another point of confusion is that there are times when the body is programmed to produce more insulin than usual and become insulin resistant on purpose to allow glucose to be redistributed in special ways, such as during puberty and during pregnancy. This is called *physiological* insulin resistance, and it is completely healthy and normal.

“**Hyperinsulinemia**” (high blood insulin) is perhaps the most accurate term for insulin resistance. Unfortunately, this term isn’t very user-friendly, but it gets right to the root of the problem, because high insulin levels are what cause insulin resistance in the first place.

Phase Three: Type 2 Diabetes (and Most Other Health Catastrophes)

Type 2 diabetes occurs when the vicious cycle of climbing insulin levels and worsening insulin resistance eventually damages your metabolism to

the degree that even persistently high levels of insulin can't bring your blood glucose back down to normal—not even after fasting all night long. As mentioned, when your fasting blood glucose rises above 125 mg/dl, you officially have type 2 diabetes.

Insulin resistance is sometimes called prediabetes because every year, 10 percent of people with insulin resistance will progress to type 2 diabetes if they don't change course¹⁰—but type 2 diabetes is just the tip of the iceberg, because persistently high insulin levels and insulin resistance secretly set the stage for most of the diseases we dread. It would therefore be just as accurate to think of insulin resistance as “pre-fatty liver,” “pre-obesity,” or “pre-heart attack.”

Insulin resistance either instigates or aggravates all of the conditions you see listed here.

Health Condition	Effect of High Insulin
Non-alcoholic fatty liver disease (NAFLD)	Insulin tells the liver to turn excess glucose into fat. ¹¹ 98% of people with NAFLD have insulin resistance. ¹²
Tinnitus, vertigo, and hearing loss	High insulin levels disrupt electrical signaling in the ear. More than 90% of people with inner ear problems such as tinnitus, vertigo, and hearing loss have high insulin levels. ¹³
Coronary artery disease (CAD)	High insulin promotes high blood pressure and inflammation of the coronary arteries; also impairs their ability to relax. ¹⁴ At least ¾ of people with CAD have insulin resistance. ¹⁵
Obesity	High insulin tells fat cells to stop burning fat. ¹⁶ More than 90% of people with obesity have insulin resistance. ¹⁷
Gallbladder disease	High insulin tells the liver to overproduce cholesterol, thickening bile. ¹⁸
Breast cancer	High insulin tells breast cells to grow and multiply more than they should. ¹⁹
Colon cancer	High insulin tells colon cells to grow and multiply more than

	they should. ²⁰
Polycystic ovarian syndrome (PCOS) and infertility	High insulin levels raise testosterone levels in the ovaries. 70% of women with PCOS have insulin resistance. ²¹
Prostate enlargement	The higher the fasting insulin, the faster the prostate grows. ²²
Erectile dysfunction (ED)	Insulin resistance impairs ability of blood vessels in the penis to relax and dilate. More than 50% of men with ED have insulin resistance. ²³
Stroke	High insulin levels promote blood clot formation and make it harder for blood vessels to relax and dilate. ²⁴
High blood pressure	Insulin tells kidneys to retain sodium and water. ²⁵ 50% of people with high blood pressure have insulin resistance. ²⁶
Acne	High insulin levels raise levels of <i>androgens</i> , hormones that tell pores to overproduce an oily/waxy substance called sebum. About ¾ of people with acne have insulin resistance; the higher the insulin level, the more severe the acne. ²⁷

Some of these conditions have become so common that we think of them as a normal part of getting older, but many of these conditions are driven more by insulin resistance than by age. Do you have any of these conditions? If so, you may have insulin resistance—and the more of these conditions you have, the more likely it is.

As you can see, insulin resistance itself is a dangerous metabolic disease, whether it ever progresses to type 2 diabetes or not, yet four out of five Americans who have this hazardous condition don't know they have it. How can that be?

Unfortunately, most doctors don't look for insulin resistance. Instead, they look for full-blown diabetes by ordering a fasting blood glucose or a hemoglobin A1C (a test that reflects your average blood glucose over the past three months). The problem with this approach is that both of these glucose tests can look perfectly normal even if you have had insulin resistance for many years. Remember: people with insulin resistance have

high insulin levels, and all that extra insulin keeps glucose levels under control... until it can't. That means that fasting glucose is the last domino to fall. We think of type 2 diabetes as a glucose problem, but it begins as an insulin problem that gradually worsens over time—it can take up to twenty years for insulin resistance to progress to type 2 diabetes.²⁸ This is why a much better test for metabolic dysfunction is a fasting insulin level. This inexpensive blood test tells you how much insulin your pancreas has to produce to keep your glucose in check.

I test all of my patients for insulin resistance—and I train other psychiatric professionals to do the same—because it provides insight into brain metabolism that a standard psychiatric interview cannot. Testing is essential because traits we think of as hallmarks of good health such as youth and physical fitness do not protect people from insulin resistance. Children, slender people, and even elite athletes can have insulin resistance, which is why so many seemingly healthy people go undiagnosed until it's too late.

We have known for decades how dangerous glucose and insulin regulation problems are for our physical health, but we have only recently begun to piece together that our mental health is just as vulnerable to these metabolic disturbances, *if not more so*, because the brain is so exquisitely sensitive to fluctuations in fuel supply.

The scientific exploration of the connection between metabolic problems and most psychiatric problems is still in its infancy, except when it comes to Alzheimer's disease. A robust body of research has produced multiple lines of high-quality evidence detailing the relationship between insulin resistance and Alzheimer's disease, and the verdict is in: Insulin resistance is a key driving force behind most cases of Alzheimer's disease.²⁹

Do You HAVE INSULIN RESISTANCE?

There is no single direct test for insulin resistance, so doctors use a combination of three simple blood tests—fasting insulin, fasting lipids (cholesterol and triglycerides), and fasting glucose—to estimate where you stand on the insulin

resistance spectrum. If you've had any of those tests in the past six months or so (and you haven't made any significant changes to your lifestyle since then), you can use the results from those tests. If not, ask your doctor to order these tests for you (or you can order tests yourself; see [chapter 17](#)). You don't need every test on the list—just choose a few that you've already had or that are easiest for you to obtain. Note: "fasting" means nothing to eat or drink except water for twelve to fourteen hours prior to the test.

Test	Result
Fasting insulin	Higher than 10 µU/ml makes insulin resistance very likely Stay in the single digits; below 6 µU/ml is ideal
Fasting blood glucose	Above 100 mg/dl = insulin resistance Below 100 mg/dl is good; between 70 and 85 mg/dL is ideal
Fasting triglycerides	Below 100 mg/dl is ideal Over 150 mg/dl makes insulin

	resistance very likely (Note: African Americans can have very low fasting triglycerides but still have insulin resistance)
HDL cholesterol	Men: higher than 40 mg/dl is good Women: higher than 50 mg/dl is good
Triglyceride-to-HDL ratio (Divide your triglycerides by your HDL)	Below 2.0 is good; the closer to 1.0 the better (Your triglycerides should be no more than twice your HDL)
Waist-to-height ratio (Divide your waist circumference by your height)	Below 0.5 is good (Your waist circumference should be less than half your height)

HOMA-IR (Homeostatic Model Assessment of Insulin Resistance)	[fasting insulin (μ U/ml) x fasting glucose (mg/dl)] \div 405 Less than 1.0 is excellent 1.8 or higher indicates insulin resistance
Kraft insulin assay This is the most accurate insulin resistance test available to consumers but also the most complicated. It measures your glucose <i>and your insulin</i> levels at several timepoints before and after drinking a 75 gram dose of glucose to see how your metabolism handles the glucose load. Learn more in this informative video by Ivor Cummins featuring Dr. Joseph R. Kraft, the pathologist who developed this test: https://youtu.be/w0nV-ddXoc .	

LEARNING FROM ALZHEIMER'S DISEASE

Alzheimer's disease robs one in ten Americans over the age of sixty-five of their dignity and humanity by slowly eroding their intellectual, emotional, and physical capabilities. More than six million Americans suffer from Alzheimer's disease today, and this number is expected to more than double by the year 2050,³⁰ taking an enormous toll on the hearts, minds, and wallets of individuals, families, and communities. And while Americans may lead the way, Americans are not alone; at fifty-five million patients and counting,³¹ Alzheimer's is now the most common neurodegenerative disease in the world.

Sadly, we are all intimately familiar with the contours of this disease. First come forgetfulness, word-finding problems, and occasional mild confusion. Increasingly, everyday tasks become challenging, and clouds of depression, irritability or suspiciousness may roll in. As the disease marches on, the ability to recognize beloved faces wanes, serious psychiatric symptoms including paranoia and even hallucinations may emerge, and bodily functions as basic as swallowing eventually become impossible. How is it that a single illness can cause such a staggering variety of disabling symptoms? Most people think of Alzheimer's as a memory disease, but in fact Alzheimer's is a neurodegenerative disease that cripples and kills not just memory-making cells, but many other brain cells as well, eventually leading to the widespread disintegration of the brain.

We feel hopeless in the face of Alzheimer's disease, because we're told that nobody knows what causes it, and that no effective treatments exist. According to the Alzheimer's Association, Alzheimer's disease is the only disease in the top ten causes of death in the United States that cannot be prevented, slowed, or cured.³² Until recently, despite thousands of clinical trials conducted over four decades and costing billions of dollars, not a single medicine had emerged that could provide meaningful relief of symptoms, let alone alter the course of the illness.³³ This dismal track record has led several major companies to scale back³⁴ or entirely abandon³⁵ their Alzheimer's drug development programs. It was against this backdrop of failure and hopelessness that the pharmaceutical company Biogen cajoled the FDA in 2021 into fast-tracking approval for a brand-new monoclonal antibody treatment for Alzheimer's disease called *aducanumab*, and a sister medication called *lecanemab* was approved in 2022. These controversial intravenous drugs cost tens of thousands of dollars per year, offer barely perceptible improvements in cognitive test scores, and come with a significant risk of brain bleeding and swelling.³⁶

Experts tell us the major risk factors for Alzheimer's disease—age, genetics, and family history—lie completely beyond our control. Messages like these make us feel like sitting ducks, simply waiting around to see whether Alzheimer's disease will strike us next.

The little-known truth is that scientists now understand a great deal about what lays the groundwork for most cases of Alzheimer's disease—more than enough to confidently conclude that this scourge may be largely

preventable.

Is Alzheimer's Disease a Modern Malady?

We can't travel back in time to study the brains of our ancestors from centuries past, but written records tell us that prior to the 1900s, descriptions of Alzheimer's and similar forms of dementia are hard to find,³⁷ suggesting that this vicious disease may be a relatively new phenomenon. In fact, the very origin of the word "dementia" dates back only to the early 1800s. In 1906, when German psychiatrist Dr. Alois Alzheimer first described the disease that bears his name, the condition was considered extremely rare.³⁸ Little did he know that a century later, his name would become a household word.

Alzheimer's disease has become so commonplace that most of us think of it as a normal part of the aging process.³⁹ But consider this: one-third of Americans over the age of eighty-five falls prey to this ruthless condition—which means that two-thirds do not.

We all know people over eighty-five with razor-sharp minds who lead rich, fulfilling lives. To this day, my ninety-year-old mother works full time caring for adults with developmental disabilities—keeping track of their appointments, medications, food preferences, social calendars, and countless other important details. She knows all the best local driving shortcuts, has a quick wit, and a zest for life. The fact that the majority of the "oldest old" members of society are spared dementia makes it plain that Alzheimer's disease is not part of the normal aging process. This then begs the question: Why is it that some brains deteriorate with age while other brains thrive?

Insulin Resistance Is Pre-Alzheimer's Disease

For decades, scientists hunted for the answer to this fundamental question. Then, in 2005, Brown University neuroscientist Dr. Suzanne de la Monte published a groundbreaking paper proposing a fresh new theory about the origins of Alzheimer's dementia: "AD [Alzheimer's disease] may represent a neuro-endocrine disorder that resembles, yet is distinct from, diabetes mellitus. Therefore, we propose the term, 'Type 3 Diabetes' to reflect this newly identified pathogenic mechanism of neurodegeneration."⁴⁰

Cited by nearly 2,000 scientific publications since, this important paper continues to inspire new studies around the world, contributing to a growing body of scientific knowledge that supports Dr. de la Monte's pioneering insights. Understanding that the destruction of our most precious organ is driven in large part by blood sugar and insulin regulation problems—*problems we already know how to manage*—represents a profound paradigm shift in the field and ushers in an exciting new era of Alzheimer's disease prevention.

A staggering 81 percent of people with Alzheimer's disease have insulin resistance or type 2 diabetes, a rate twice as high as among their cognitively healthy peers.⁴¹ Furthermore, the younger you are when you are diagnosed with type 2 diabetes, the greater your risk of developing Alzheimer's as you age.⁴² The term "type 3 diabetes" is catching on because it summarizes in three words the powerful connection between two of the most common and most dreaded diseases we face: type 2 diabetes and Alzheimer's disease—both of which are driven by insulin resistance.

Insulin Resistance and the Brain

Recall that brain glucose levels mirror blood glucose levels, so the higher your blood sugar, the higher your brain sugar. With insulin and the brain, it's a different story.

In those of us with persistently high blood insulin levels, the receptors responsible for escorting insulin across the blood-brain barrier can become increasingly resistant to insulin, making it harder and harder for insulin to penetrate the brain.⁴³ Therefore, over time, as counterintuitive as it sounds: The higher your blood insulin, the *lower* your brain insulin.

Low brain insulin is a serious problem, because without insulin, brain cells can't process glucose and turn it into the energy and components they need to thrive. Cells deprived of adequate insulin can't utilize glucose at full capacity; so instead of flourishing, they sputter and struggle to maintain normal operations. This dire predicament is called *cerebral glucose hypometabolism*—sluggish brain glucose processing. This brain energy slowdown can be detected with a PET scan (positron emission tomography scan)—a technique that generates three-dimensional images of the brain's glucose usage patterns. (These scans cost thousands of dollars and are not

usually covered by insurance.) PET scan studies conclusively show that the more insulin resistant you are, the less glucose your brain can utilize.⁴⁴ Your brain will still absorb plenty of glucose, but it will become increasingly difficult for it to absorb enough insulin to process that glucose. If your brain has become insulin resistant, it can be swimming in a sea of glucose and still be starving to death.

The Hungry Hippocampus. Which brain cells are among the first to suffer the consequences of an insulin shortage? The cells of the hippocampus—your brain’s learning and memory center. The hippocampus is a seahorse-shaped mini-organ buried deep inside your brain with special properties that make it particularly vulnerable to insulin resistance.

Whenever you’re learning something for the first time, memory cells inside the hippocampus sprout tiny new dendritic spines to connect with neighboring cells and build new circuits. (You may recall that this remarkable ability of the brain to remodel itself in response to our experiences is called neuroplasticity.) As a master growth hormone, insulin is crucial to the process of dendritic spine formation, growth, and survival.⁴⁵ The hippocampus also possesses the rare ability to give birth to brand-new brain cells⁴⁶—yet another growth process that requires insulin.

Hippocampal cells need so much energy to do their important work that they often require extra boosts of glucose. These special glucose surges require insulin, making the hippocampus even more prone to problems than other areas of the brain when insulin is in short supply.⁴⁷ If there isn’t enough insulin in your brain to supercharge your hippocampus, or if your hippocampus isn’t responding normally to insulin’s signals, recording new memories will be challenging. This helps to explain why short-term memory problems are among the earliest signs of this disease; for people with Alzheimer’s dementia, learning new information or recalling recent events is much harder than remembering things that happened a long time ago.

Without access to adequate insulin, the hungry hippocampus struggles to chronicle new memories, and its cells begin to shrivel and die. *Hippocampal atrophy* (shrinkage of the hippocampus) is an ominous harbinger of Alzheimer’s disease. By the time a person headed for Alzheimer’s disease notices memory problems, the hippocampus has already shrunk by more than 10 percent.⁴⁸

Plaques and Tangles: Smoking Guns or Red Herrings? Sadly, the fundamental role insulin resistance plays in the development of Alzheimer's disease remains underappreciated. Instead, many experts, institutions, and organizations continue to devote much of their time and resources to flashier elements of the disorder: amyloid plaques and neurofibrillary tangles.

This fixation began in 1906, when a woman by the name of Auguste Deter died in a hospital in Frankfurt, Germany, after suffering for five long years with severe, progressive paranoia and confusion. The origin of her symptoms being a mystery, the hospital director sent brain specimens from Frau Deter's autopsy to Dr. Alzheimer in Munich for examination.⁴⁹ Dr. Alzheimer was a neuropathology expert who had spent many years studying and writing about brain anatomy, so when he scrutinized Frau Deter's brain under the microscope and saw the two peculiar protein formations that we now refer to as amyloid plaques and neurofibrillary tangles, he knew these were new and important discoveries.

In the long hunt for treatable root causes of dementia, these plaques and tangles have remained the focus of intense scientific curiosity and research, and many investigators continue to believe these oddities drive the development of dementia. As you'll see, while they may play some role in the course of the disease, it's becoming clear they are not in the driver's seat.⁵⁰

Amyloid Plaques

Under stress, brain cells release protein fragments called amyloid peptides to help defend themselves—so amyloid itself is good. However, if these fragments clump together into amyloid plaques, they can interfere with brain cell signaling, but they do not appear to be the root cause of Alzheimer's disease. Forty percent of people over age eighty are walking around with amyloid plaques in their brains yet have not a whiff of cognitive impairment.⁵¹ Furthermore, researchers have spent decades of time and billions of dollars studying amyloid and designing drugs to attack it, yet, with the dubious exception of the new monoclonal antibody treatments, every single one of these drugs has failed in clinical trials.⁵²

It appears that the insulin-resistant brain may be fertile ground for

plaque development. As a hormone intimately involved in growth and repair, insulin stimulates production of insulin-degrading enzyme—which not only breaks down used insulin molecules but helps clear away excess amyloid molecules as well. People with low brain insulin therefore have lower levels of this enzyme, making it easier for excess amyloid to clump into plaques. In this way, maintaining healthy levels of brain insulin may help protect against plaque formation in the first place.⁵³

Neurofibrillary Tangles

Some scientists who have lost faith in the amyloid hypothesis have turned their attention to the tantalizing possibility that neurofibrillary tangles might be more to blame for the brain's descent into dementia. Neurofibrillary tangles are also big abnormal clumps of proteins—tau proteins, to be exact. Tau is a housekeeping molecule that helps maintain cell infrastructure. When tau proteins abandon their posts, they can band together and form large, dysfunctional, flame-shaped tangles that accumulate inside cells, disrupting normal brain cell architecture. Tau tangles can wreak such havoc that some cells may ultimately decide to commit *apoptosis*—cellular suicide.⁵⁴

People are more likely to experience cognitive problems if they have tangles than if they have plaques,⁵⁵ so it is understandable that some scientists find them attractive as potential Alzheimer's disease culprits. But again, before we become obsessed with tangle removal, it is worth stopping first to wonder: What prompts tau proteins to gang up on the very cells they are supposed to be taking care of?

In the healthy brain, insulin works to keep tau proteins at their stations, maintaining order. However, when insulin is in short supply, tau proteins desert their stations and accumulate into toxic heaps. This is just one more way in which insulin deficiency in the brain helps set the stage for neurodegeneration and cell death.⁵⁶

Guilt by Association? The tau hypothesis has taken off like a rocket, but some scientists caution that the tau theory may be yet another red herring distracting us from the true underlying causes of Alzheimer's disease. It is beginning to look like the tale of tau may mirror not only amyloid's story, but also the stories of other well-meaning proteins unfairly blamed for the

neurodegenerative diseases they happen to be associated with. Alzheimer's disease, Parkinson's disease, Huntington's disease, and Lou Gehrig's disease (ALS) all sport curious clumps of signature proteins that have captured the imagination of pharmaceutical researchers, luring them into careers spent aiming their talents at the wrong targets.

The human body is intelligently designed—everything it does, it does for a reason. Does the aging brain purposefully churn out toxic dysfunctional proteins to hasten its own demise? Unlikely. In a 2019 review of the evidence, scientists from New Zealand's University of Canterbury Mental Health and Nutrition Research Group pointed to problems with brain metabolism as the most likely driver of plaques, tangles, and the progression of Alzheimer's disease.⁵⁷

Neurotransmitter Problems: Acetylcholine and Glutamate

In addition to filling up with plaques and tangles, the Alzheimer's brain suffers from imbalances in neurotransmitters important to memory function. One hallmark of Alzheimer's disease is the gradual disappearance of brain cells that produce acetylcholine. When this problem was first identified in the 1970s, it was considered a revolutionary breakthrough in Alzheimer's research and led to the development of drugs designed to prolong the activity of the dwindling amounts of acetylcholine the brain was still able to produce. However, these medicines—donepezil (Aricept), rivastigmine (Exelon), and galantamine (Razadyne)—can't bring back the cells that have already vanished, and they are powerless to prevent the ongoing destruction of the acetylcholine-producing cells that remain. What is killing these precious cells? Tau and amyloid accumulation are partly to blame, but low brain insulin activity helps pull the plug by reducing the supply of *nerve growth factor*—a nurturing protein these cells need in order to thrive.⁵⁸

The other commonly prescribed drug that may provide some support for the Alzheimer's brain is memantine (Namenda), a medicine that tones down the activity of glutamate, a neurotransmitter that can spin out of control in Alzheimer's disease. The process of neuroplasticity requires fine tuning of glutamate to get the levels just right. Too little glutamate shifts the brain into neutral, bringing construction and maintenance of new synapses to a halt, but too much glutamate shifts the brain into a toxic state of overdrive,

fueling the inflammation and oxidative stress that directly contribute to the widespread neurodegeneration we see in Alzheimer's disease.⁵⁹ Insulin resistance contributes to this problem both by interfering with healthy glutamate metabolism and function⁶⁰ and by promoting amyloid and tau buildup; amyloid plaques and tau tangles can damage the glutamate regulation system over time and allow glutamate to reach dangerously high levels that are capable of triggering cell death.⁶¹

Are You Already on the Road to Alzheimer's Disease?

Just as type 2 diabetes doesn't happen overnight, neither does Alzheimer's disease—Alzheimer's disease is quietly preceded by *decades* of dwindling brain glucose metabolism.⁶² Sluggish brain glucose processing has been documented in people in their fifties, sixties, and seventies who have no cognitive symptoms at all.⁶³ In fact, your brain can lose up to a quarter of its total glucose-processing power before you notice any problems with your memory.⁶⁴ Researchers have even found PET scan evidence of brain glucose processing problems in insulin-resistant women in their *early twenties* who also reported no cognitive issues.⁶⁵ Yet deep inside their brains, unbeknownst to them, glucose processing is slowing down, plaques and tangles are accumulating, neurons are malfunctioning, and the hippocampus is silently shrinking. The realization that dementia develops so stealthily prompted the National Institute on Aging and the Alzheimer's Association to create a new diagnostic system in 2011 that now includes two new pre-clinical (symptom-free) stages which can only be detected by brain imaging (PET scan, MRI) or lumbar puncture (spinal tap),⁶⁶ but these are expensive, invasive tests that are not available to most people.

Fortunately, there is a much simpler and more empowering tool at your disposal that can provide insight into your brain's metabolism: you can evaluate yourself for insulin resistance.

Just as you can't afford to wait until your fasting glucose becomes abnormal to think about type 2 diabetes, you can't afford to wait until you reach a certain age or notice memory problems to think about Alzheimer's. The good news is that this slow, downhill slide in brain power has much more to do with your metabolic health than with your age, your amyloid levels, or your ancestry,⁶⁷ meaning you have the power to influence the

course of your intellectual future by changing your diet.

IS INSULIN RESISTANCE “PRE-MENTAL ILLNESS?”

Decades of careful Alzheimer’s research has taught us this: The fundamental problem that insulin resistance creates for the brain is restricted access to energy. We still have a lot to learn about how insulin resistance influences psychiatric conditions other than Alzheimer’s disease, but it stands to reason that an energy shortage could make it more difficult for the brain to control mood, thinking, and behavior, and emerging evidence supports this idea.

Signs of insulin resistance are strongly associated with psychiatric disorders; that is to say, people with insulin resistance are much more likely to suffer poor mental health, and vice versa. For example:

- People with glucose levels in the prediabetes range are 2.7 times more likely to develop major depression than people with normal glucose levels.⁶⁸
- People with newly diagnosed bipolar disorder are 3.5 times more likely to have metabolic syndrome than their peers without bipolar disorder.⁶⁹
- People with newly diagnosed schizophrenia are 3.7 times more likely to have insulin resistance than their peers without schizophrenia.⁷⁰

Did you notice the size of these associations? You may recall from [chapter 3](#) that for an association between any two things to be meaningful, it should be at least 2.0. These associations all fall above this threshold. They still can’t tell us whether metabolic problems *cause* these mental health conditions—but they are strong enough to tell us that the relationship between the two may be more than just a coincidence—a causal relationship between the two is *plausible*. Given everything Alzheimer’s research has taught us about how insulin resistance disrupts normal brain structure and function, it would be surprising to me if insulin resistance *didn’t* have the potential to cause other mental health disorders—or at least to play a significant role in their origin or severity.

People with Alzheimer's disease often suffer from symptoms that look identical to those of major mental illnesses such as depression, mood swings, anxiety, paranoia, hallucinations, and insomnia. When symptoms such as these appear in young people without Alzheimer's, could they be early warning signs of brain degeneration? In other words, could brain energy deficits be the root cause or at least a contributing cause of psychiatric disorders other than Alzheimer's disease?

We've known for a very long time that depression, bipolar disorder, and schizophrenia are chronic illnesses that can worsen over the years, and even lead to cognitive decline. PET scans of depressed patients who don't respond to antidepressants show pronounced slowing in brain glucose processing when compared to patients who improve on antidepressants.⁷¹ Furthermore, a large multinational study⁷² found that people with depression had on average a 24 percent reduction in the size of the hippocampus—the same brain region notorious for shrinking in Alzheimer's disease—which may help to explain why depression *doubles* your risk for developing dementia later in life.⁷³ Stanford University psychiatrist Dr. Natalie Rasgon was the first to notice that insulin resistance could be “the missing link” between these two common conditions, writing in 2005: “Insulin resistance may be an important therapeutic target for effective management of depressive disorders, and possibly, prevention of Alzheimer's Disease.”⁷⁴

In bipolar disorder, some brain areas process glucose better than others, but overall, there is a reduction in glucose usage compared to people without bipolar disorder.⁷⁵ Hippocampus size is also smaller,⁷⁶ and having a diagnosis of bipolar disorder more than doubles your chances of developing dementia later in life.⁷⁷

In people with schizophrenia who also have insulin resistance, brain cells process glucose more slowly, causing glucose to accumulate to higher levels inside the brain. This group of people also performs less well on memory tests than those without insulin resistance.⁷⁸ Hippocampus size is also smaller in people with schizophrenia,⁷⁹ and people with schizophrenia are 2.5 times more likely to be diagnosed with dementia as they age.⁸⁰

Researchers have also detected sluggish brain glucose processing in adults who have had ADHD (attention deficit hyperactivity disorder) since childhood,⁸¹ people with borderline personality disorder,⁸² and people with

OCD (obsessive-compulsive disorder) who have hoarding behaviors.⁸³ Given that insulin resistance and compromised brain glucose processing are features of so many different psychiatric conditions, it would make sense to offer patients treatment options capable of addressing these head-on.

Beyond Association: Insulin Resistance and Bipolar Disorder

Dr. Cynthia Calkin, a metabolic psychiatrist at Dalhousie University, is the first researcher to put this to the test in a randomized controlled trial. In 2015, she observed that her patients with bipolar disorder who also happened to have insulin resistance or type 2 diabetes were more likely to have chronic mood symptoms, more likely to experience rapid cycling mood patterns, and less likely to respond to lithium, a mood-stabilizing medication.⁸⁴ She then designed a sophisticated experimental protocol to explore whether reversing insulin resistance using metformin (a medicine commonly prescribed for type 2 diabetes) could help people with longstanding bipolar disorder who had tried multiple psychiatric medications without relief. The results of her study, published in 2022, were striking: reversal of insulin resistance *substantially* improved depression symptoms in *every* case.⁸⁵

When I asked Dr. Calkin what it felt like to witness these outcomes, she said, “Almost 90% of the patients in [this] study had not had a single remission in 25 years. It has been a relief to discover a new mechanism-based treatment that can get these patients better.”⁸⁶

In an interview with the CBC (Canadian Broadcasting Corporation), Kellie Williams, a Nova Scotia resident who participated in the study, shared her experience:⁸⁷ “My depression felt like if someone had passed away.... There were some times, several times, where I contemplated suicide. To try and get rid of that despair or pain I felt, I actually started cutting as well, just to try and redirect that pain you feel inside your head.” After reversal of her insulin resistance—a condition she didn’t know she had until Dr. Calkin tested her for it—her depression went away. “I couldn’t believe it was actually gone.” Williams added, “I’d never felt such a sense of wellness in my life. This is truly a miracle.”

If reversing insulin resistance with a simple diabetes medication helps people with bipolar disorder feel better, it would be reasonable to think that

reversing insulin resistance with lifestyle changes could do the same. One of the most effective ways to address insulin resistance is with a ketogenic diet, and in the next chapter, you will see how much promise this intervention holds not only for the treatment of bipolar disorder, but for the treatment of many other psychiatric conditions as well.

Piecing the Puzzle Together

If you have insulin resistance, and you eat in an unhealthy way, your brain glucose levels will be unstable, your brain insulin activity will be low, and your brain will struggle to produce energy. Excess glucose sugarcoats vital brain molecules, setting off waves of inflammation and oxidative stress that your low-insulin brain lacks the power and materials to fight.⁸⁸ Without the resources it needs to neutralize oxidative stress and heal inflammation, your brain's fragile landscape could continue to smolder indefinitely.

How exactly will chronic inflammation, oxidative stress, and sluggish brain glucose processing affect *your* mental health? I believe this is where genetics, family history, early life nutrition, environmental exposures, and life experiences come into play. Whether you develop mood, concentration, anxiety, social, behavioral, or memory problems—or some combination of these—may depend largely on the biological cards you have been dealt and how you have lived your life to this point. But take heart: No matter what your past looks like or what your vulnerabilities may be, you have it within your power to fundamentally change the way your brain does business, and that could make all the difference.

CHAPTER 9

The Promise of Ketogenic Diets for Mental Health

I'm convinced that nutritional measures can have a profound impact and lead to very important benefits, which makes me say that this represents the future direction to develop in psychiatric care.

—Albert Danan, MD

Several years ago, a young man with epilepsy and autism behaviors adopted a ketogenic diet and experienced substantial improvements in both conditions within only a few weeks, ultimately becoming seizure-free. It just so happened that this young man was a relative of my friend and colleague Dr. Albert Danan, who has been practicing psychiatry in Toulouse, France, for more than thirty-five years. Having witnessed this remarkable transformation in brain health in a member of his own family, Dr. Danan became curious about whether this same diet might be able to help some of his own patients.

The population Dr. Danan serves is comprised primarily of people of French and North African descent with severe mental illnesses, most of whom he has been working closely with for many years or even decades. He invited thirty-one of his most treatment-resistant patients with major depression, bipolar disorder, or *schizoaffective disorder* (a form of schizophrenia which also affects mood) to try a ketogenic diet under his close supervision in the Clinique du Castelvieu, a local psychiatric hospital. As is so often the case in people with chronic mental illness, all of these volunteers also had markers of poor metabolic health such as high blood glucose, high blood pressure, high triglycerides, and obesity. Many were unable to work due to psychiatric disability. All of them were taking

multiple psychiatric medications, and all of them had been hospitalized before by Dr. Danan one or more times at either the Clinique du Castelvieu or at a similar sister hospital nearby. Having exhausted all other treatment options, they agreed to participate.

What happened was extraordinary.

By week three, every one of the volunteers who stuck with the plan (twenty-eight of the original thirty-one) began improving—both metabolically and psychiatrically. All twenty-three people with depression symptoms experienced substantial improvements in mood, and all ten people with schizoaffective disorder experienced substantial reductions in psychosis symptoms. Twelve people (44 percent) achieved full clinical remission, and eighteen people were discharged from the hospital on less psychiatric medication than they were taking at the time of admission. All but one patient lost weight, despite the fact that nearly all were taking antipsychotic medications, which are notorious for causing stubborn weight gain.

The dietary prescription used in this study was adapted from the protocol that one of our co-authors, Dr. Eric Westman, has used in his weight-loss research at Duke University for many years. The plan consisted almost exclusively of meat, seafood, poultry, eggs, vegetables, nuts, and cheese; it was low in carbohydrate (20 grams per day maximum) and moderate in protein (15 to 20 percent of calories), with the remainder of daily calorie requirements coming from fat. The diet was well tolerated, and no medical or psychiatric safety issues arose.

DR. DANAN'S DIET PROTOCOL

15 to 20 percent protein, 75 to 80 percent fat, and 5 percent carbohydrate

- Protein: Meat, seafood, poultry, and eggs—including their natural fats, free of starchy coating. (Limit to 15–20 percent of daily calories.)
- Eat 2 cups of salad vegetables per day. Permitted

vegetables include all leafy greens, arugula, bok choy, cabbage, chives, endive, radishes, scallions, and watercress.

- Eat 1 cup of cooked vegetables per day (measured cooked). Permitted vegetables include artichokes, asparagus, broccoli, Brussels sprouts, cauliflower, celery, cucumber, eggplant, green beans, jicama, leeks, mushrooms, okra, olives, onions, peppers, pumpkin, shallots, snow peas, bean sprouts, alfalfa sprouts, sugar snap peas, summer squash, tomatoes, rhubarb, wax beans, and zucchini.
 - Permitted snack foods: ham, pâté, chorizo, salami, hard-boiled eggs, sugar-free gelatin.
 - Other permitted foods: cheese, up to 100 g per day; cream and oils, 2 to 8 tablespoons per day (depending on weight loss goals); mayonnaise, 2 to 3 tablespoons per day; lemon juice, up to 4 teaspoons per day; avocado, up to 1 per day; 85% dark chocolate, 2 squares per day; soy sauce, up to 2 tablespoons per day.
 - Drink plenty of (sugar-free) fluids when thirsty, and consume broth twice a day.
-

These outcomes were so unexpected that Dr. Danan and I collaborated with Duke University obesity medicine researcher Dr. Eric Westman and University of Michigan behavioral medicine researcher Dr. Laura Saslow to publish them in June of 2022.¹

As a co-author of this paper, naturally I view its findings in a positive light, but having practiced psychiatry for more than twenty years I can tell you that we never see results like this with standard psychiatric treatments. Clinical remission is rare, most people leave psychiatric hospitals on more medication, and metabolic side effects such as weight gain are common. In this case, the remission rate was high, and instead of side effects, people

were enjoying *side benefits*: healthy reductions in weight, blood pressure, blood sugar, liver enzymes, and triglycerides.

This wasn't a randomized controlled trial, so we can't be certain that the ketogenic diet itself was responsible for the unprecedented improvements we reported. However, we believe it contributed considerably to these outcomes, as they would otherwise be difficult to explain. These patients had previously benefited very little from attentive outpatient care, multiple medications, and psychiatric hospitalization—the only difference between this hospitalization and previous hospitalizations was the addition of the ketogenic diet.

These observations offer tremendous hope, because they suggest that a ketogenic diet could bring significant relief to people with serious mental illnesses, regardless of the nature or duration of their symptoms.

WHAT IS THE KETOGENIC DIET?

The ketogenic diet is any way of eating that lowers insulin levels enough to burn fat and generate metabolically meaningful levels of ketones in the blood.

Most experts consider metabolically meaningful ketosis to begin at blood ketone levels of 0.5 mM (millimolar). I'll show you how to measure ketones in [chapter 18](#), but for now, just know that if your blood ketone level is at least 0.5 mM, you are "in ketosis."

There is no one-size-fits-all ketogenic diet plan because we are each metabolically different, and we have different dietary preferences. Some people think a ketogenic diet must be high in animal protein, contain lots of saturated fat, or completely exclude fruit, but these impressions are inaccurate. The ketogenic diet isn't a dietary pattern; so long as you achieve ketosis, you can follow any dietary pattern you like—including vegan (no animal foods), carnivore (no plant foods), and everything in between. There are even strategies such as calorie restriction and fasting that will shift your metabolism into ketosis while still allowing a fair amount of carbohydrate, but continuing strategies like those long-term would be unsustainable, so the ketogenic diets in this book will follow these widely accepted guidelines:

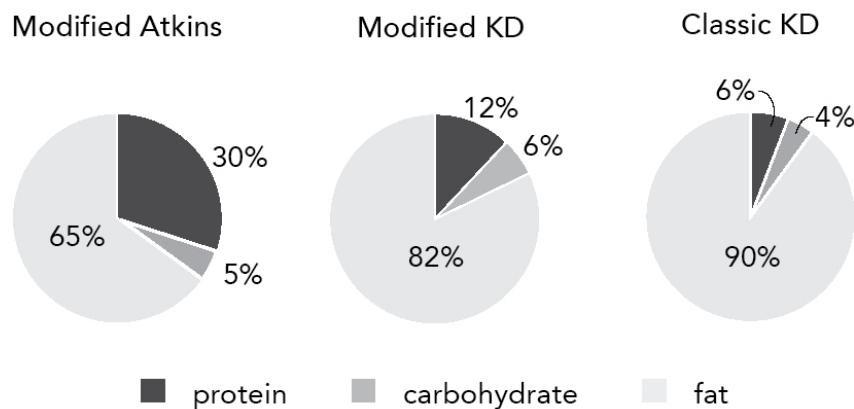
Ketogenic diets should be very low in carbohydrate. The goal of any

ketogenic diet is to lower insulin. Since carbohydrate is the macronutrient that tends to raise insulin the most, lowering carbohydrate is key; from a metabolic perspective, carbohydrate is the “noisiest” macronutrient you can eat.

Ketogenic diets should be moderate in protein. Overeating protein can interfere with ketosis because protein stimulates insulin—although, in most cases, not nearly as much as carbohydrate does. However, protein is essential and it is the most important macronutrient in your diet, so undereating protein can be dangerous.

Ketogenic diets should be relatively high in fat. The majority of calories in ketogenic diets come from fat instead of carbohydrate. Fat stimulates almost no insulin response, so from a metabolic perspective, fat is the “quietest” macronutrient you can eat.

Ketogenic diets exist on a spectrum from the relaxed *modified Atkins diet* shown above on the left, to the strict *classic ketogenic diet* on the right. (The original Atkins diet, created more than fifty years ago by cardiologist Dr. Robert Atkins for weight loss, allowed for a gradual increase in carbohydrate over time and placed no limit on protein or fat, so it wasn’t necessarily ketogenic.)



KETOGENIC DIET VARIATIONS

- The modified Atkins diet caps carbohydrate at 20 grams per day and allows for liberal amounts of protein.
- The classic ketogenic diet, created by Dr. Russell Wilder in 1921 to treat children with epilepsy, places tight limits on both protein and

carbohydrate.

- In between these two extremes are modified ketogenic diets, which allow for moderate amounts of protein.

All three of these diets are very low in carbohydrate but they differ in the amount of protein they allow. Notice that as you move from left to right, protein goes down, which lowers insulin to a greater and greater degree. As protein decreases, fat increases to take its place. Combining carbohydrate restriction with protein restriction lowers insulin the most and raises ketones more robustly, making diets on the right more ketogenic than diets on the left. Think of protein + carbohydrate as a ketone control knob: the less protein + carbohydrate in the diet, the higher ketones will tend to be. I'll show you how to personalize your macronutrients in [chapter 18](#), but in most cases, lowering carbohydrate to about 20 grams per day is a good place to start.

How Does the Ketogenic Diet Improve Brain Health?

It may surprise you to hear that we know more about how ketogenic diets affect the brain than we know about psychiatric medications, because scientists and clinicians have been using and studying the ketogenic diet since 1921, decades before any psychiatric medication had been developed. Unlike medications, which target just one or maybe a few brain chemistry problems at most (such as the activity of a particular neurotransmitter), the ketogenic diet is a *multi-purpose tool* that addresses numerous underlying problems simultaneously:

- Cools inflammation²
- Bolsters antioxidant defenses³
- Partially bypasses defects in damaged mitochondria⁴
- Repairs and replenishes mitochondria⁵
- Supports neuroplasticity (creation of new brain circuits)⁶
- Resolves *sensory gating* deficits that cause hallucinations⁷
- Rebalances glutamate, GABA, and other neurotransmitter systems⁸
- Stabilizes brain cell networks⁹

- Lowers sodium levels inside neurons, making them less reactive¹⁰
- Bridges the brain glucose energy gap¹¹

In short, the ketogenic diet stabilizes, protects, and energizes the whole brain, helping to restore peaceful equilibrium across multiple systems.

By keeping your carbohydrate intake low, you also keep your blood glucose levels low and stable—no spikes or crashes in your bloodstream, therefore no spikes or crashes in your brain either. This alone goes a long way toward stabilizing brain chemistry. Lowering your brain glucose levels makes it less likely that you will produce advanced glycation end products (AGEs)—those sticky, dysfunctional molecules we discussed in [chapter 6](#) that promote inflammation and oxidative stress throughout the brain.

By keeping your insulin levels low, you allow your ketones to rise into the therapeutic range. If you have a history of insulin resistance (which most of us do), then your brain has trouble burning glucose for energy. Ketones cross easily into the brain and help bridge that energy gap, which many of my patients experience as a wonderful and welcome feeling. One gentleman in his seventies told me, “I feel like my brain just woke up from a long winter’s nap.” Phrases like “mental clarity,” and “calm, focused energy” are ones I hear frequently. When I first tried a ketogenic diet myself back in 2012, I wrote in my food journal, “This diet is peaceful.”

I call my diet plans “Quiet Diets,” not only because they have the power to bring peace of mind, but also because that power lies not in the presence of special ingredients like superfoods or supplements, but in the absence of everyday ingredients that overstimulate, overwhelm, and aggravate our biology. Simplifying your diet by removing substances that interfere with your body’s wisely designed systems allows your biochemical pathways to function at their best.

IS THE KETOGENIC DIET SAFE?

Most concerns about the ketogenic diet stem from the three ways in which these diets differ from those recommended by mainstream nutrition authorities:

Criticism #1: The ketogenic diet is dangerously low in carbohydrate. The USDA and many other nutrition authorities recommend we eat a diet containing 45–65 percent carbohydrate, despite the fact that the human body can make all the glucose it needs from fat and protein. Given that most of us have lost our ability to safely process large amounts of carbohydrate, one could argue that the USDA diet is dangerously *high* in carbohydrate. As UK-based nutrition researcher Dr. Zoe Harcombe testified to the British Parliament in 2019: “Don’t base guidelines on the one macronutrient that we don’t need and that diabetics can’t handle.”¹²

Criticism #2: The ketogenic diet is dangerously high in fat. The ketogenic diet essentially replaces most carbohydrate calories with fat calories, bringing the fat percentage up to 70 percent or more. This worries those who believe that eating fat makes us fat, but you already know that insulin is what tells your body to store fat, and carbohydrate turns insulin up. This also worries those who believe that eating fat is dangerous because it raises cholesterol levels, clogs arteries, and causes heart disease, but you already know that insulin resistance is what sets the stage for inflammation and constriction of the coronary arteries. I’ll address the cholesterol concerns in [chapter 11](#), but for now, suffice it to say that the *type* of fat you eat is far more important than the *amount* of fat you eat. Public health enemy number one is not high-fat diets, it is high-insulin diets, and fat is the macronutrient that raises insulin the least.

Criticism #3: Being in ketosis is dangerous. Ketosis is a normal, natural, healing state that satisfies the brain’s energy needs whenever we are not eating carbohydrates. Ketosis is not to be confused with diabetic ketoacidosis, which is a medically dangerous state of runaway ketone production that only occurs in cases of severe insulin

deficiency (such as type 1 diabetes) or when taking medications that increase the risk for ketoacidosis.

The bottom line is that ketogenic diets are safe for most adults, provided they are properly constructed of nutritious whole foods, contain appropriate amounts of protein, that simple steps are taken in the beginning to minimize common discomforts that can occur as your body adapts to the diet, and that all medications and existing medical and psychiatric conditions are closely monitored by your health care team.

THE KETOGENIC DIET FOR SPECIFIC PSYCHIATRIC CONDITIONS

The exciting, emerging field of metabolic psychiatry hasn't yet produced large, randomized controlled trials of the ketogenic diet for psychiatric conditions, but scientific interest in this area is exploding. At the time of this writing, there are clinical trials in the pipeline for anxiety, bipolar disorder, psychosis, substance abuse, post-traumatic stress disorder, college mental health, and Alzheimer's disease. In the meantime, in addition to Dr. Danan's work, we have plenty of small studies, published case reports, and patient testimonials to educate and inspire us. If you have been living with any of these mental health conditions, following a ketogenic diet such as the plan I offer in this book may bring you much-needed relief. However, the ketogenic diet is a powerful metabolic intervention that requires careful planning and medical monitoring, so if you are considering this approach, please read [chapter 18](#) carefully first to learn how to use this strategy safely.

Bipolar Disorder

There is no clearer example of brain energy regulation gone haywire than bipolar disorder.

People with bipolar disorder have sudden, unpredictable shifts in mood that include manic episodes—unusually high-energy states that make it extremely difficult to sleep and may also include euphoria, grandiosity,

racing thoughts, pressured speech, hyperactivity, and other signs of increased energy. If manic episodes are severe, the illness is called bipolar I disorder; if they are mild (*hypomania*), the illness is called bipolar II disorder. Stretches of mania or hypomania are often followed by deep, severe depressions that can feel physically and intellectually paralyzing.

One of the many problems with brain metabolism that can occur in bipolar disorder is difficulty transferring glucose fragments to the electron transport chain in Engine M where they can be efficiently churned into large amounts of energy. As a result, brain cells are left to rely mainly on glycolysis (Engine G, the simple glucose-chopping engine) to burn glucose, but glycolysis alone simply can't generate enough power to support the hardworking sodium-potassium pumps needed to keep neurons primed and ready to fire. If energy supply is unreliable, those pumps will malfunction, causing neurons to either fire too much (mania) or too little (depression).¹³ You can think of this as a brain brownout: without a steady supply of energy, neurons will flicker on and off at unpredictable times.

There are three published case reports of women with bipolar disorder who found the ketogenic diet so helpful that they were able to successfully discontinue all mood stabilizing medications,¹⁴ and formal clinical trials are already under way. Stanford University metabolic psychiatrist Dr. Shebani Sethi is conducting the first ever clinical trial of ketogenic diets in bipolar disorder, expected to conclude in 2023. The second trial is being led by a team of scientists at the University of Edinburgh that includes Dr. Iain Campbell, who put his own bipolar disorder into long-term remission using a ketogenic diet.

KETOGENIC DIET PUTS BIPOLAR II DISORDER INTO REMISSION

Seven years ago, a talented Scottish musician with bipolar disorder tried a ketogenic diet for weight loss. Dr. Iain Campbell is now one of the world's leading researchers in the field of metabolic psychiatry, specializing in the study of ketogenic diets for bipolar disorder.

With bipolar 2, the depressive episodes are the worst part for me. At my worst, I'd experience long and unyielding depression for weeks and months that were physically and mentally exhausting—performing even normal, daily tasks was a struggle.

At some point, I decided if I was going to be depressed for the rest of my life, I could at least be at a healthy weight. My diet was a typical SAD, including plenty of bread, cereal, pasta, and sugar. I noticed that around the time the depressions became more severe for me I was putting on weight much more easily. So I tried a low fat diet, but I felt terrible and found it impossible to sustainably lose weight. I learned about the Atkins diet and heard it worked well for weight loss, so I adhered to it strictly. My focus was weight loss; I hadn't heard about any benefits for mental health but the plan, along with regular exercise, put me into intermittent ketosis. I started to notice a definite shift in my mental well-being.

The first time, I was riding the bus on my way to work, and I became aware that I was experiencing something fundamentally different in my mind state. For the first time in 15 years, I felt the lights in my brain switch back on... it felt like I had a TV with the color contrast and brightness turned way down to where everything looked flat and dim. Now I was seeing bright colors rippling across the screen in subtle waves of positive emotion in response to normal daily events. I had no idea people generally experienced this in their day-to-day life.

I had tried almost everything you could imagine over the years—medications, CBT, meditation, therapy, intense exercise, cold exposure, heat exposure, binaural beats, breathing techniques, countless supplements, vitamins, minerals, philosophy, travel, Camus, Kerouac, Nietzsche (briefly)—you name it! I

found small benefits in other approaches, but never true relief. Going into ketosis felt like switching on a backup power generator for my brain that I never knew I needed. It felt like it was addressing the root cause of my condition vs. making small improvements in symptoms.

When I experienced the mood stabilisation and did some research, I learned about ketosis and so began to follow a full ketogenic diet. I tracked my ketones and mood around every 3 days over the course of a year and noticed that I got the best results when above 2mM. When I am in established ketosis above 2 mM my mood becomes stable, I have consistent energy, I sleep better, and I have a much clearer mind. Benefits seem to increase the longer I stay in ketosis.

I have been in ketosis the majority of the last 7 years. When I keep to a regular schedule with good sleep and exercise, I find it easy to maintain the diet over long periods. The times it becomes more difficult are when a significant disruption occurs beyond my control, like heavily disrupted sleep which can lead to food cravings. During these times I have to have strategies to deal with it (such as pre-preparing keto snacks). My adherence is strongly motivated by wanting to stay well, and I get immediate feedback, mostly in the form of depression, if I diverge from the diet even for a day. My thinking grinds to a slower pace and my productivity drops dramatically. I can't make decisions, and everything feels overwhelming to me.

The available literature indicates that bipolar disorder is as much a condition of energy dysregulation as it is a mood disorder, and there are now decades of research providing evidence of disruption of mitochondrial function and glucose metabolism in the brain. Ketones act as an alternative energy substrate for mitochondria in the brain, which may act to stabilise brain energy

metabolism when glucose metabolism is compromised. Ketosis promotes stable energy in the brain while reducing hyperexcitability and protecting neurons from damage. If you were to design a treatment for bipolar disorder it would be hard to think of a better combination of effects.

When my symptoms of bipolar became noticeably worse in my teenage years, I also noticed significant weight gain, which was previously unusual for me. I think this indicated the onset of a systemic metabolic dysfunction. Bipolar is definitely a genetic condition but environmental factors can trigger the onset of illness. I think it's likely I would have experienced symptoms regardless, but perhaps if I had been aware of the problems with refined carbohydrates, I could have been well for longer and avoided more damage.

I will need to stay on the diet permanently to not experience symptoms, so ketosis hasn't cured me. But it is an excellent treatment that allows me a quality of life I never would have experienced without it. I feel a responsibility to those with bipolar to let them know that this may be an option for them. I only wish I could have communicated this to people with bipolar in the dark ages of insulin shock therapy and lobotomy.

The genes for bipolar are well preserved in the population and I think that there are adaptive traits and rare mental capacities people with bipolar can use to contribute to the world. However, these same capacities make them particularly vulnerable to our unnatural modern environment. I believe this perspective is important to move the focus of treatment away from simple sedation to treatments which create an environment where people with bipolar can thrive.

—Iain Campbell, PhD, Baszucki Research Fellow in Metabolic Psychiatry, Division of Psychiatry, University

Major Depression

In 2022, Virta Health, an innovative virtual diabetes care clinic based in the United States, published a study that included thirty-six patients with mild clinical depression. After following a modified Atkins diet for ten weeks, more than half of the depressed patients no longer met criteria for depression. The average blood ketone level of all participants was 0.6 mM, and those who spent more days in ketosis (0.5 mM or above) were more likely to experience mood benefits.¹⁵ One of the ways that ketogenic diets may help with depression is by cooling inflammation. For example, ketosis has been shown to shift microglia (the brain's immune surveillance cells) from a pro-inflammatory state to an anti-inflammatory state.¹⁶

KETOGENIC DIET DISSOLVES DECADES OF DEPRESSION AND ANXIETY

Eric had a happy home life and excelled in school, but by third grade he had become anxious and was teased for being overweight. He ate sugary cereals for breakfast and snacked on junk food after school. His mom cooked wholesome dinners, but these were always followed by dessert, and he snacked again before bed; by senior year of high school, he weighed 215 pounds. He lost twenty pounds before college by limiting himself to three meals a day, but was “still eating junk,” still had some excess belly fat, and despite also taking up running, he developed high blood pressure in his mid-twenties.

Eric’s first bout of depression occurred at age thirty and was mild, but his second episode, which occurred seven years later, after his father passed away, was much more serious: He made a plan to commit suicide, even circling the

date on his calendar. He said, “If I didn’t feel better by then, that would be it.” His doctor prescribed sleep medication, which did help, but some degree of depression persisted, and the obsessive anxiety he’d had since childhood was “building like water coming to a boil,” so at age forty, he started running marathons.

“The running was treatment for me, and it helped, but I had to do a lot of it. I would feel horrible all day long, but then at seven p.m. I would run three miles and would start thinking to myself ‘I don’t want to die right now,’ but by bedtime that good feeling was gone again.”

At age forty-five he saw a psychiatrist who provided counseling and prescribed Lexapro (an antidepressant), both of which helped somewhat until age fifty-five when, after changing jobs, he experienced his deepest depression yet. “I had thoughts of suicide for most days for months on end.” He had no energy to exercise, so his weight rose to 205 pounds. He was referred to a psychiatrist, and recalls asking him: “I have a son in college, a good marriage, and my wife and I make good money—so why do I want to die?”

After several years of antidepressant changes, he arrived at Effexor, which thankfully eased his depression, but also caused side effects. So, on the advice of a popular book, he adopted a vegan diet and started running again. He lost weight, and his mood improved somewhat, but the diet caused significant gastrointestinal distress, and it didn’t completely resolve his depression, so he discontinued it after about six months.

At age fifty-six, he started a low-carbohydrate diet, and felt so much better that he was able to slowly taper off all psychiatric medication. His triglycerides dropped from 170 mg/dl to 65 mg/dl, his hemoglobin A1C is normal at 5.4 percent, and at six feet two inches tall, his weight is a healthy 170 pounds. He’s been following a low-carbohydrate diet for more than six years and remains symptom-free.

“I have become a better version of me than I have been for

my entire adult life. It's ridiculous how good I feel. It's a miracle, really. I wouldn't believe it if it didn't happen to me. I absolutely have a new life. I realize diet cannot fix every mental problem, but it 100 percent fixed mine."

Schizophrenia

The first report describing the use of the ketogenic diet to treat mental illness was published in 1965. Clinicians at the Central Louisiana State Hospital placed ten hospitalized women who were being treated for schizophrenia on a ketogenic diet for two weeks and noted significant improvement.¹⁷ The field then lay silent until 2009, when a seventy-year-old woman who weighed 330 pounds was referred to Dr. Eric Westman's obesity clinic at Duke University. She also happened to have schizophrenia. Despite taking two antipsychotics, an antidepressant, and a sleep medication, she was plagued by chronic, disabling symptoms including paranoia, suicidal thoughts, voices in her head, and had been experiencing visual hallucinations of skeletons daily since the age of seven. Dr. Westman prescribed a simple modified Atkins diet for weight loss and within eight days her psychotic symptoms began to improve. She was soon able to stop all psychiatric medications, and over time she lost 150 pounds. At the core of this powerful antipsychotic therapy was a simple modified Atkins diet of beef, chicken, turkey, ham, fish, green beans, tomatoes, sugar-free beverages, and water. Years later, her psychotic symptoms are still in remission.¹⁸

Since that seminal 2009 case report, five additional case reports have documented substantial improvements in schizophrenia symptoms in response to ketogenic diets,¹⁹ and three clinical trials of ketogenic diets for schizophrenia are under way at Stanford University, University of California, San Francisco, and James Cook University in North Queensland, Australia.

There are many ways in which a ketogenic diet may address root causes of schizophrenia symptoms. In addition to inflammation, oxidative stress, and brain brownout—problems which schizophrenia shares with bipolar disorder—people with hallucinations can have *sensory gating deficits*,

meaning that the brain's natural ability to filter out and ignore irrelevant sounds doesn't work properly. In animal studies, the ketogenic diet completely restores healthy sensory gating mechanisms.²⁰

Autism

Autism spectrum conditions, which occur in approximately 1 percent of children worldwide,²¹ are neurodevelopmental disorders that affect how people behave and interact with others and are usually noticeable by age two. Two published case reports and three small clinical trials of ketogenic diets point to a variety of potential benefits for children with these developmental differences. One of the studies was a six-month randomized controlled trial conducted in Egypt that divided forty-five children into three groups: a balanced diet, a gluten-free/casein-free diet (casein is a dairy protein), and a ketogenic diet. Behavioral symptoms improved most in the gluten-free/casein-free group, whereas social, speech, and cognitive symptoms improved most on a ketogenic diet. The balanced diet resulted in no benefits.

A common problem seen in autism is that mitochondria are less efficient at turning glucose into energy partly because of a specific defect in complex I, the main gateway to the electron transport chain. Ketogenic diets partially bypass that defective area by feeding more fuel molecules directly into complex II, allowing damaged mitochondria to generate energy efficiently again.²² Ketogenic diets also improve the health of existing mitochondria and stimulate the production of new mitochondria.

Binge Eating and Food Addiction

Binge eating and food addiction are common concerns among my patients and cause a great deal of distress, a sense of powerlessness, and loss of self-esteem, not to mention significant weight gain. I view these conditions less as psychiatric disorders and more as the predictable result of ultraprocessed foods specifically engineered to be addictive, but regardless of the underlying cause, I have found the ketogenic diet to be a powerful tool for addressing these concerns.

In a published case series, three people with binge eating disorder, food addiction symptoms, and obesity responded beautifully to a modified

Atkins diet that limited carbohydrate to a maximum of 30 grams per day, bringing binge episodes down from once or twice per day to nearly zero episodes per week. Six months later, their symptoms were still in remission, and they had lost 10 to 24 percent of their body weight.²³

Patient	Binge episodes per week at start	Binge episodes per week after 6 months	Weight loss
54-year-old woman	14+	0	37 lbs
34-year-old man	8-11	0	44 lbs
62-year-old woman	8-10	0	22 lbs

Ketogenic diets can help with food addiction and binge eating by stabilizing glucose and insulin levels, which in turn stabilizes hunger hormones and neurotransmitter systems.²⁴ By reliably fueling cells in between meals, ketogenic diets reduce cravings and make it easier to go for longer periods of time without thinking about food.

KETOGENIC DIET RAPIDLY REVERSES FOOD AND MOOD ISSUES

Deanna (not her real name) is a sixty-four-year-old woman I consulted with who had lived with food addiction, binge eating, depression, and anxiety for forty years. Her usual pattern was that she would begin snacking at 4:00 p.m. and then binge all evening long. She told me: “I know it’s chemical in my brain,” “My brain feels hijacked,” “I’ve gained so much weight.”

She had tried many medications to try to suppress her appetite in the past, including Prozac (an antidepressant), Vyvanse (a stimulant), topiramate (a seizure medication that can reduce appetite), and naltrexone (a medicine that can

reduce cravings for addictive substances by blocking opioid receptors in the brain).

Before changing her diet, I asked her to write down all of her symptoms:

- Very irritated
- Depressed
- Nonproductive
- Brain fog
- Want to sleep all day
- Hopeless
- Want to isolate
- Don't want to move my body
- Bowels messed up
- Aching joints
- Gaining weight
- No life at all

Deanna met full criteria for food addiction, scoring 11 out of 11 on the modified Yale Food Addiction Scale (mY-FAS-2.0), full criteria for generalized anxiety disorder, scoring 19 out of 21 on the Generalized Anxiety Disorder scale (GAD-7), and full criteria for depression as measured by both the Patient Health Questionnaire (PHQ-9) and the Beck Depression Inventory (BDI), on which her score fell into the severe depression range. (All of these diagnostic scales are available to you in appendix A should you wish to evaluate yourself for these conditions.)

She was too depressed and overwhelmed to follow a ketogenic diet (which requires counting carbohydrates), so I asked her to start simple by eating only meat, seafood, poultry, and eggs without counting or tracking anything for the first week or two to get the ball rolling. Within four days, her blood ketone level was 2.1 mM, and by day twelve, she no

longer met criteria for any psychiatric diagnosis.

Assessment Scale	Day Zero	12 Days Later
mY-FAS-2.0 (food addiction)	11/11	1/11
GAD-7 (anxiety)	19/21	0/21
PHQ-9 (depression)	23/27	0/27
BDI (depression)	37/63 (severe)	7/63 (normal)

She wrote: "After just one week of eating no carbs there is a dramatic change, emotionally, physically, my mood, and outlook on life. All of my symptoms become just the opposite!!!! I become less irritated, and the brain fog lifts. My joints don't ache, my mood improves every day. No cravings for sweets. I just need to look at that list every day and remind myself that I don't want to go back!"

Substance Use Disorders

In a small randomized controlled trial, patients hospitalized for the management of alcohol withdrawal who were placed on a strict classic ketogenic diet experienced fewer alcohol cravings and required about 50 percent less oxazepam, a medication commonly prescribed to keep people safe and comfortable as they transition to abstinence.²⁵ Blood ketone levels in this study exceeded 4.0 mM.

Alcohol stimulates the activity of GABA, the brain's calming "brake pedal" neurotransmitter, so when people who have become physically dependent on alcohol stop drinking, GABA activity suddenly drops, upsetting the balance between GABA and glutamate (the brain's stimulatory "gas pedal" neurotransmitter). One of the ways ketogenic diets may help with alcohol withdrawal is by promoting a healthier balance between glutamate and GABA; ketosis supports higher GABA activity, helping to fill the GABA gap created by the discontinuation of alcohol.

Alzheimer's Disease

The exciting possibility that ketones may help rescue starving brain cells has sparked dozens of studies of Alzheimer's disease in the past decade alone, including several human clinical trials. However, given that Alzheimer's is an advanced neurodegenerative disease, it is much more difficult to treat. Some studies use ketogenic diets alone, while others use supplements like ketone salts or medium-chain triglycerides (MCTs)—oils that the body converts quickly into ketones. (There is more information about supplements in [chapter 18](#).) We'll focus mainly on the diet studies here.

A 2012 study conducted at the University of Cincinnati found that people with mild cognitive impairment (pre-Alzheimer's) who lowered their carbohydrate intake to about 35 grams per day scored a little better on cognitive tests than those eating a standard high-carbohydrate diet.²⁶ They also had lower blood glucose and insulin levels, naturally ate many fewer calories, and lost more weight.

A 2017 study conducted at the University of Kansas found that people with early Alzheimer's disease who lowered their carbohydrate intake to about 45 grams per day and took MCT oil saw improvements on cognitive tests, and while the improvements were small, they were as good if not better than we typically see with Alzheimer's drugs like Aricept.²⁷

The most rigorous trial to date was a 2021 RCT conducted by researchers in New Zealand in which a ketogenic diet was compared to a low-fat diet in patients with Alzheimer's disease.²⁸ In this study, patients achieved higher ketone levels on average (just under 1.0 mM) than in other studies, and while they did score higher on tests of daily function and quality of life, their cognitive test scores did not improve.

If ketones are supposed to help bridge the brain energy gap seen in Alzheimer's disease, why don't we see a more robust cognitive response to the ketogenic diet in studies such as these? Perhaps higher ketone levels would have led to better outcomes, but since Alzheimer's disease is a neurodegenerative disease in which a significant degree of damage has already taken place by the time symptoms become apparent, the sobering truth is that some of that damage may not be reversible. It is therefore important to identify and address metabolic dysfunction as early as

possible.

THE KETOGENIC DIET TURNS A BRAIN AROUND

Fran is a retired registered dietitian living in Colorado. About twenty years ago, at age forty-four, she slipped on the ice. Her head struck the ground, causing a neck injury and a concussion. She soon developed seizures and started struggling to remember things. She'd forget dates of important events such as upcoming family weddings, hunt for hours for her car in parking lots, and get lost on the way to important work obligations. She was diagnosed with mild cognitive impairment, and was prescribed lamotrigine (Lamictal), an anticonvulsant medication, to control seizures.

Her cognitive abilities appeared to hold steady until her sixties, when she began noticing a bit more difficulty with memory and occasional trouble finding her words, but the changes were too subtle for her primary care physician (PCP) to detect on a cognitive screening test.

Then, at age 67, something scary happened. As Fran tells it:

In April of 2021, my life was falling apart. After sewing for a lifetime I could no longer figure out how to thread my machine. With both diagnoses of epilepsy and mild cognitive impairment twenty years ago, I knew there was a good chance I was progressing into a dementia. I saw my PCP about my condition. I scored miserably enough [on a cognitive test] for him to highly suspect that I had Alzheimer's-type dementia, and he literally told me to go and enjoy the rest of my life. This was devastating. My cognition continued to get worse, and I didn't want to live that way.

When her son reached out to me for advice, I offered to consult with her.

Fran continues:

My son was familiar with the utility of ketogenic diets for brain health, and we knew there was little promise in much else, so I started the ketogenic diet and implemented it to a T. And over time, I improved. I saw a neurologist who was thrilled that I was on the diet. He tested me while on the diet, and I scored 27 out of 28 on his test. He said, "It's time to take this tentative diagnosis off of your chart."

I do know I was in serious trouble. Entering ketosis dramatically improved my condition to the point that I regained my ability to sew, to think more clearly, and to know that I can live like tomorrow is going to be great.

At the time of this writing, Fran has been faithfully following a ketogenic diet for twenty-two months and maintains blood ketone levels at around 0.7 mM. She changed her diet as follows:

Old Diet:

- Breakfast: Raisin Bran and skim milk, toast and butter
- Lunch: burger or hotdog on bun, Diet Coke
- Dinner: pasta meal with meat sauce, Italian bread, salad
- Snacks: crackers and cheese, peanuts, bread and butter

New Diet:

- Breakfast: none (fasts from bedtime until noon)
- Late morning: coconut oil in beef broth
- Late lunch: large salad (romaine, egg, tuna, cheese, nuts, bacon) with homemade keto poppyseed dressing
- Dinner: cheese, bacon, and mushroom quesadilla on low-

carb sun-dried tomato tortilla

- Beverage: diet tonic water with lime
- Evening: coconut oil in beef broth
- Snacks: pecans sautéed in lots of butter with erythritol brown sugar; keto ice cream bar

After a lifetime of enjoying brownies made from scratch and home-baked artisanal breads, Fran is now thriving on a brownie-free, bread-free diet. When I asked her if she missed baking bread or having pasta for dinner, she became emotional and shook her head no. “I sat in front of the sewing machine and couldn’t thread it. I sat talking with my son and couldn’t finish my sentences. I was scared to death. I believe I have the commitment to do this for the rest of my life.”

THE FINE PRINT

Published cases and clinical trials don’t yet exist for other psychiatric diagnoses such as attention deficit disorder, generalized anxiety disorder, post-traumatic stress disorder, and obsessive-compulsive disorder. However, since these are all brain-based conditions, and the ketogenic diet improves multiple aspects of brain health, there is reason to believe it has the potential to be helpful with all of them.

As inspiring as these stories are, the ketogenic diet is not a mental health cure-all. However, in my clinical experience, most people with psychiatric conditions do benefit, and those who are able to follow the diet longer term are often able to reduce the number and/or dosage of psychiatric medications they take. There are also some who benefit from a ketogenic diet but continue to need full medication support.

However, medication reduction isn’t the only measure of success. Some of my patients have no interest in discontinuing medication because it allowed them to stay in school, kept them out of the hospital, or saved their life. Patients like these don’t want to go off medication, they just want to feel better and be healthier *on* medication. One of the many side benefits of

the ketogenic diet is that it can counteract some of the most common and concerning side effects of psychiatric medications, such as drowsiness, high blood glucose levels, high triglycerides, and weight gain. Even if the ketogenic diet doesn't make a big difference in your mental health, improving your metabolic health is a major victory in and of itself.

There are certain situations in which ketogenic diets should not be used, and precautions to be aware of (see [chapter 18](#)), but by and large, the ketogenic diet is a low-risk, high-potential-benefit intervention well worth considering in the management of most mental health issues from minor everyday concerns such as anxiety, irritability, and brain fog to major mental illnesses. This is especially good news for people whose symptoms don't fall neatly into any diagnostic category.

Beyond Ketosis

Ketogenic diets support and stabilize brain metabolism with ketones, allowing the brain to operate more cleanly, smoothly, and efficiently, but optimal mental health is about more than just ketones. The nutritional quality of your diet matters, too. If it's optimal brain health you seek, you'll need to do more than just energize your brain; you'll need to nourish and protect it as well, and this is where food choices come into play. Building your ketogenic diet from whole foods is key—but one of the best-kept secrets in nutrition science is that just because a food is whole doesn't necessarily make it healthy. So which whole foods are best at nourishing and protecting your brain?

PART 3

THE WHOLE TRUTH ABOUT WHOLE FOODS

CHAPTER 10

Meat: The Original “Superfood”

Red meat is not bad for you. Now blue-green meat, that's bad for you!

—Tommy Smothers

Meat is good for you.

These five words represent a simple statement of biological fact. While there remain many unanswered questions about nutrition, the question of whether animal foods belong in the human diet is not one of them. Meat provides all of the macronutrients and micronutrients we need, in their proper forms—including some that are difficult or impossible to obtain from plant foods. Unlike plant foods, meat contains no substances that interfere with our ability to absorb or utilize nutrients. Meat is easy to digest and supports healthy insulin levels without promoting blood glucose spikes. The human brain evolved to require animal-source foods and therefore cannot develop or function properly without them.

In a perfect world, this would be all anyone needs to know about meat—but given the widespread efforts to blame meat for everything from constipation to cardiovascular disease to cancer, it is not enough to explain the biological benefits of meat and let that information speak for itself. Alarming headlines proclaiming that meat—especially red meat—can harm us or even kill us demand our attention, therefore I devote the lion's share of this chapter to the dismantling of popular anti-meat arguments so that you can see them for the paper tigers they are. All that said, we cannot turn a blind eye to the harsh realities of industrialized meat production systems. While I am convinced by the science that meat belongs in a brain-healthy diet, I am equally convinced that the way many animal foods are produced is inhumane and unhealthy for animals, humans, and the planet, so we will

wrestle with these problems as well, in search of a better way forward.

Before we begin, please note that throughout this chapter, I will use the word “meat” to refer not only to “red meat” (mammal meats such as beef, lamb, pork, venison, and bison), but to all types of animal meat, including seafood, poultry, and organ meats such as liver.

WHAT IS RED MEAT?

Let’s start with the basics. What is the difference between red meat and white meat? Red meat simply contains more *heme iron*, which happens to be red. We have been taught to think of all mammal meat as red meat and all poultry meat as white meat, but this is biologically incorrect. All animals, including fish and birds, need heme iron, therefore all animals contain at least some red meat.

Heme is a ring-shaped protein with a hole in the middle to hold a metal ion. When the metal in the middle happens to be an iron atom, the molecule is called “heme iron.” Heme iron empowers the hemoglobin in blood to carry oxygen around the body, the myoglobin in muscles to store oxygen for exercise, and the mitochondria in cells to usher electrons through the electron transport chain, helping turn food molecules into energy. Hardworking “slow-twitch” muscles, particularly those used for endurance—including muscles needed to simply stand up all day (think cow)—need lots of oxygen to sustain their activity, so they are richer in heme and therefore redder in color (although in sedentary or very young animals, these muscles will be more pink than red). “Fast-twitch” muscles that power short bursts of activity are used less often and contain less heme, so they’re paler in color. Most fish don’t need strong muscles because water supports their weight, so their flesh usually isn’t red except in their fins and tails, but fish that swim fast for long distances such as tuna and sharks have more red meat. The breasts and wings of chickens and turkeys are “light meat” because they don’t fly much, but their legs and thighs are “dark meat” because they stand and walk on them all day. The meat of nearly all other birds including ducks, geese, and ostriches is red; in fact, ostrich meat contains just as much heme iron as roast beef.¹

Are Humans Supposed to Eat Meat?

Human beings are omnivores, meaning we are biologically equipped to consume both plant and animal foods. It is impossible to know precisely which foods our prehistoric ancestors consumed and how often, but logical to assume that they chose from whatever plants and animals were seasonally available in their geographic location. For example, humans living near the equator would likely have had year-round access to abundant plant foods such as tropical fruits and starchy root vegetables, as well as a great diversity of mammals, birds, reptiles, insects, and aqueous creatures, whereas humans living near the North Pole would have had year-round access to Arctic mammals, birds, and ocean creatures, but little to no opportunity to consume plant foods of any kind for most of the year.

While there is passionate debate about how much meat our prehistoric ancestors ate, most paleontologists agree that meat was an indispensable part of their diets.² Chimpanzees, our closest primate relatives, supplement their mostly plant diet with insects and small amounts of meat. Does this mean that we humans could also thrive on a mostly plant diet?

Our prehistoric ancestors began evolving away from chimpanzee ancestors more than six million years ago.³ The earliest evidence that pre-humans used tools to prepare meat dates back more than three million years, and evidence that meat had become an important staple food dates back two million years.⁴ It was at around this same time that our ancestors' abdominal cavities began to shrink, likely reflecting the shortening of the digestive tract.⁵ Extracting nutrients from plant foods is a slow process that requires the assistance of bacterial fermentation in the lower intestine, whereas meat is efficiently digested by stomach acid and gut enzymes, then absorbed in the upper intestine. As a result of eating more meat and fewer plants, our overall gastrointestinal tract became smaller than that of chimpanzees; our colons are less than half the size, and our small intestines nearly twice as long.⁶ The human brain eventually grew to become three times larger than the chimpanzee's, and its cerebral cortex (the layer responsible for complex thought) contains twice as many cells.⁷

Some scientists argue that eating meat made us human—meaning that meat allowed us to devote less energy and bodily real estate to the long intestinal tract needed to process high-fiber, high-plant diets, so that we

could invest more energy in developing our uniquely oversized brains.⁸

MEAT IS NUTRIENT RICH

As our ancestors' diets shifted to include more meat, it became much easier to provide the brain with the macronutrients and micronutrients it needs, particularly in early childhood, when the brain is rapidly growing and developing but the digestive system is still rather small and immature.⁹ This is because, contrary to popular belief, meat contains more nutrients per ounce than plant foods and is easier to digest.

Macronutrients

Protein: Animal foods provide the highest quality protein available. All meat proteins are considered *complete* proteins because they contain adequate amounts of all nine essential amino acids.

Fat: Animals store most of their energy as fat, so all meats contain fat, whereas plants store most of their energy as starch, so most plant foods contain less fat than most animal foods. All animal fats naturally contain a mixture of saturated and monounsaturated fat, as well as vital polyunsaturated fatty acids (PUFAs), including the omega-6 arachidonic acid, and the omega-3 fatty acids EPA and DHA. DHA is particularly precious because it's very difficult for us to make from plant fats, and because it plays indispensable roles in brain development, brain energy production, and brain cell communication.

Carbohydrate: All non-dairy animal foods are naturally extremely low in carbohydrate, but they are not carbohydrate-free. For example, nearly all cells contain DNA and RNA, which contain sugar backbones; all cell membranes contain fatty molecules with sugary components (glycoproteins and glycolipids); and muscle and liver cells contain glycogen (liver contains one gram of carbohydrate per ounce). Nevertheless, the amount of detectable carbohydrate in non-dairy animal foods is extremely low compared to plant foods, and nutrition labels tend to list the carbohydrate content of most non-dairy animal foods as 0 grams per serving.

Cholesterol: Since all animal cells need cholesterol, all animal foods contain cholesterol (plant cells need cholesterol, too, but they use a different

kind). However, there are certain animal foods that are particularly high in cholesterol. Liver is rich in cholesterol because the liver is where most of the body's cholesterol is manufactured. Glandular organ meats (pancreas, kidney, sweetbreads, etc.) are higher in cholesterol because glands produce hormones, which are made from cholesterol. The brain houses 20 percent of the body's cholesterol because the myelin sheaths which insulate its electrical circuits are full of cholesterol. The cholesterol in milk is there because the growing baby calf needs it to build new cells, and egg yolks contain concentrated cholesterol because the growing baby chick needs it to build new cells. We'll explore the controversy surrounding cholesterol in the next chapter but suffice it to say for now that cholesterol is good, and human life would be impossible without it.

Micronutrients

We think of colorful fruits and vegetables as nutritional powerhouses teeming with vitamins and minerals, but a little-known nutrition science fact is this: *Just because a food contains a nutrient doesn't necessarily mean our bodies can access it.* Many plant nutrients suffer from poor bioavailability—meaning that they come in forms that are harder for us to use, or that naturally occurring substances within plants called *antinutrients* interfere with our ability to extract, absorb, and/or utilize them.¹⁰ For these reasons, while certain fruits and vegetables can be excellent sources of vitamin C, vitamin E, vitamin K1, and folate (vitamin B9), meat is a superior source of all other essential nutrients, including several nutrients that plants don't contain at all.

Surprisingly, it is theoretically possible to obtain all of the vitamins and minerals we need from animal-source foods alone, particularly if some organ meats are included in the diet. For example, liver is an excellent source of vitamins A, D3, E, K1, K2, and folate, which are harder to obtain from muscle meats (particularly if they are lean). Animal foods are low in vitamin C compared to many fruits and vegetables, but they do contain enough to meet our daily requirements if not overcooked. In fact, there is enough vitamin C in fresh meat to treat and prevent scurvy.¹¹ The most challenging nutrient to secure from (non-dairy) animal foods is calcium, since most of it resides in bones, shells, bone marrow, and blood, which

most people do not consume.¹²

Only animal foods contain true vitamin A (retinol), which is at least twelve times more bioavailable than the beta-carotene in plants.¹³ Meat is an excellent source of every B vitamin, including B7 (which plants contain very little of) and B12, which plants do not contain at all. Vitamin D3 from animal foods is easier to use and store than D2, the form found in mushrooms and yeast. Only animal-source foods contain the MK-4 form of vitamin K2, which is easier to absorb (and is the form used by the human brain),¹⁴ and only animal foods contain the long-chain polyunsaturated fatty acids EPA, DHA, and arachidonic acid.

Micronutrient Availability in Plant and Animal Foods

Vitamin A	12 to 24 times more bioavailable in animal foods ¹⁵
Vitamins B1, B2, B3, B6, B7	Easier to find in animal foods
Vitamin B9 (Folate)	Insoluble fiber matrix in some plant foods hinders bioavailability
Vitamin B12	Not found in plant foods
Vitamin C	Easier to find in plant foods
Vitamin D	D3 from animal foods easier to use/store than D2 from fungi and yeast
Vitamin E	Easier to find in plant foods
Vitamin K1	Easier to find in plant foods
Vitamin K2	Not found in plant foods (except in a few fermented foods, e.g., natto)
Iron	Bioavailability of heme iron (15–35%) is greater than non-heme iron (2–20%). ¹⁶ Eggs, dairy, and many plants contain compounds that interfere with iron absorption
Calcium	Some plants contain compounds that interfere with calcium absorption
Iodine	Many plants contain goitrogens that interfere with iodine utilization

Zinc	Many plants contain compounds that interfere with zinc absorption
EPA and DHA	<p>Not found in plant foods. Plant foods contain ALA, which must be converted to EPA and DHA</p> <p>Conversion of ALA to EPA is low: 8% in men and up to 21% in women¹⁷</p> <p>Conversion of ALA to DHA is very low: 0-4% in men and 9% in women¹⁸</p>

Meat contains all the nutrients we need, in their most bioavailable forms, without interference from antinutrients. The brain evolved to require meat, and meat allowed the brain to evolve. Our digestive system is engineered to process meat. How, then, has this nutritious whole food that we've been consuming since time immemorial come to be viewed as responsible for the meteoric rise in chronic physical and mental illnesses that began only in the past century or so? Why are we told that animal foods in general, and meat in particular—especially red meat—endanger our life and health?

OUR COMPLICATED RELATIONSHIP WITH MEAT

Our uniquely sophisticated cerebral cortex blesses us not only with exceptional intelligence but also with a conscience. Our ability to empathize with other species can make it psychologically uncomfortable to kill and eat other creatures, particularly those with whom we most closely identify—our fellow mammals. This spiritual connection to other mammals may help to explain why red meat in particular is so often the focus of anti-meat sentiment. Are some species more precious than others, and if so, where do we draw the line? Author Lierre Keith, who followed a vegan diet for nearly twenty years and suffered serious mental and physical health consequences as a result, wrestles with this question in *The Vegetarian Myth*. From a colony of ants unintentionally crushed while tending her organic garden to the billions of creatures in the nourishing topsoil below who would go hungry without natural fertilizer, she realized that growing her own food involves the lives and deaths of countless others.

We are dependent on a million different creatures, most of

them invisible to our eyes, all of them doing the work of producing or degrading that we cannot do... for someone to live, someone else does indeed have to die. To reject one is to reject the other because there is no way out.... Where was I going to draw the line? That was the question, my personal, political, spiritual agony. Mammals, fish, insects, plants, plankton, bacteria?... I have my answer, finally. I'm not going to draw a line. I'm going to draw a circle... [19](#)

However, our inescapable dependence on the death of others doesn't mean we can't work harder to minimize unnecessary destruction and improve the quality of their lives.

The industrialization of our food supply, which began in the mid-1800s, led to the mass production of animal foods at the expense of animal and environmental health and welfare, raising important ethical concerns that understandably have turned many away from meat. Another consequence of the Industrial Revolution was urbanization; migrating to cities physically and emotionally separated a great many of us from the land and animals that feed us. However, vegetarianism in certain circles existed long before industrialization, dating back more than 5,000 years to ancient Egypt, where it was believed that abstaining from meat would improve chances of reincarnation.[20](#) Western monastic traditions forbade the consumption of four-legged animals, Hinduism holds cows sacred, and Buddhism teaches nonviolence toward all animals. These philosophies are grounded in moral or spiritual beliefs, and some of them remain influential even today within these religious communities.

It wasn't until the 1800s that health-related arguments about the biological benefits of eating plants and the medical dangers of eating animals first appeared in the narrative. Interestingly, this line of reasoning originated not from scientific or medical institutions but from Western religious ideologies. According to research conducted by Belinda Fettke, an expert in the history of the origins of plant-based dietary trends, it was the Temperance Health Reformer movement that began sowing the seeds of vegetarianism in the Western Hemisphere.[21](#)

Historian Margaret Puskar-Pasweicz described the Temperance Health

Reformers of the early 1800s as “blending religion, science, philosophy, and politics to establish a scientific rationale for vegetarianism.”²² The cause was soon taken up by the fledgling Seventh Day Adventist Church, most notably by its co-founder Ellen G. White, who reportedly experienced more than two thousand visions from God during her lifetime, many of which informed her dietary teachings.

The Seventh Day Adventist Church, through studies it conducts at Loma Linda University and practices it promotes through the American College of Lifestyle Medicine, has since become a powerful voice in the science and politics of nutrition, influencing research and policy worldwide.²³

Unlike spiritual arguments, which tended to influence only certain segments of the population, fear of chronic disease transcends religion and culture and therefore proved to be a more effective way to change hearts and minds. Even the medical and scientific communities have been moved by these baseless claims, as evidenced by the tremendous resources they have invested in studying the potential connection between meat and declining public health. If modern scientific inquiry in this field has been largely driven by a religious hypothesis rather than a biological hypothesis, it helps us understand why hundreds of research papers attempting to find evidence that meat endangers our health have been written... and why they repeatedly come up empty-handed.

DISMANTLING ANTI-MEAT ARGUMENTS

When I advise patients to include some meat in their diets to nourish their brains, their most common concern is that it will be bad for their hearts or that it will cause cancer. Rest assured that what is good for the brain is good for the heart, and vice versa. Remember: it would not make sense for each of your organs to require a different diet; all of your cells require the same basic nutritional care.

Does Red Meat Cause Heart Disease?

The idea that red meat could cause heart disease is based primarily on two bodies of research:

1. Nutrition epidemiology studies seeking associations between red meat consumption and heart disease risk
2. Randomized controlled trials (RCTs) exploring how red meat affects blood levels of LDL (the so-called “bad cholesterol”) and/or triglycerides (fat in the bloodstream)

As for the first category of research, we’ve already discussed how nutrition epidemiology studies are unhelpful, but it may further put your mind at ease to show you how trivial the epidemiological associations between red meat and heart disease tend to be. Researchers at Cornell University and Northwestern University pooled six epidemiological studies totaling nearly 30,000 people and published a meta-analysis of their findings in the *Journal of the American Medical Association* in 2020.²⁴ They determined that eating two additional four-ounce servings of unprocessed red meat per week was associated with an increased risk for cardiovascular disease of only three percentage points, reporting this increased risk as a “hazard ratio” (which you can think of as the relative risk) of 1.03. (We learned in [chapter 3](#) that a relative risk of less than 2.0 is considered too small to be meaningful, and a relative risk of 1.0 represents no increase in risk at all.)

As for randomized controlled trials, there has actually never been a human RCT designed to explore whether eating red meat causes or worsens heart disease, perhaps in part because conducting such a study would be logistically very difficult. Instead, we have dozens of RCTs exploring whether eating red meat raises LDL cholesterol and triglycerides, because higher LDL and triglyceride levels have long been thought to increase risk for cardiovascular disease. These studies have yielded inconsistent results, with findings pointing weakly in different directions.²⁵

Despite the fact that studies haven’t yet been able to demonstrate a connection between red meat and heart disease, many researchers continue to believe it exists—even when the ingredient within red meat thought to be most dangerous for the heart—saturated fat—has been exonerated.

The Saturated Fat Hypothesis

For decades, we were told that beef was dangerous because the saturated fat it contains was supposed to drive our blood cholesterol levels up, force cholesterol into our coronary artery walls, and cause heart attacks. Fortunately, this hypothesis is dying a long overdue death. In 2020, the *Journal of the American College of Cardiology* published a state-of-the-art review of saturated fats and health, concluding that “there is no robust evidence that current population-wide arbitrary upper limits on saturated fat consumption in the United States will prevent CVD [cardiovascular disease] or reduce mortality.”²⁶

In 2021, an international panel of experts—which included two scientists who had previously served on the U.S. Dietary Guidelines Advisory Committee (DGAC)—reviewed the evidence and came to a similar conclusion: “Multiple reviews of the evidence have demonstrated that a recommendation to limit consumption of saturated fats to no more than 10 percent of total calories is not supported by rigorous scientific studies.”²⁷

Even though the saturated fat hypothesis is melting away under the heat of scientific scrutiny, the belief that red meat causes cardiovascular disease continues to beat on in the hearts of many researchers, leading some to start pointing the finger at a different molecule within red meat: *carnitine*.

The Carnitine Hypothesis

Be forewarned that the hypothetical line connecting carnitine to heart disease involves taking a long and winding detour through a completely different molecule called TMAO, so we’ll need to familiarize ourselves with both of these molecules first before we try to understand the convoluted argument against carnitine.

Carnitine is a vital nutrient that transports fat molecules into the mitochondria to be burned for energy; it is so important that if you don’t eat enough of it, your body goes out of its way to make it from scratch. Red meat contains far more carnitine than white meat because red muscle fibers use more energy. Plant foods happen to be extremely low in carnitine because they primarily burn carbohydrate for energy, not fat.

Most of the carnitine in meat is absorbed by the small intestine. If any carnitine remains unabsorbed, it will work its way down to the colon, where

certain gut bacteria can turn it into a foul-smelling gas called TMA (trimethylamine). TMA wafts into the bloodstream and enters the liver, which converts it into TMAO (trimethylamine oxide), an odorless substance that is safely eliminated in the urine.

In a 2013 study²⁸ that has since been cited in more than 2,500 scientific papers, a group of Cleveland Clinic researchers reported that carnitine promotes atherosclerosis (hardening of the arteries) by demonstrating the following:

1. Extremely high doses of L-carnitine (a poorly absorbed form of carnitine not found in meat), caused atherosclerosis in mice (that were genetically altered to easily develop atherosclerosis).
2. When human subjects were fed steak plus moderate amounts of this same form of (poorly absorbed) carnitine, some of them produced TMAO in their urine.
3. When the aforementioned atherosclerosis-prone mice (who are missing a gene required for normal cholesterol processing) were fed TMAO supplements, they excreted less cholesterol in their cage droppings, suggesting that TMAO had trapped some cholesterol in the body. This led researchers to theorize that TMAO could lead to heart disease.

Note that these experiments do not show that carnitine, TMAO, or red meat cause heart disease in humans. They don't even show that steak alone causes high TMAO levels in mice or humans, because no steak-only experiments were conducted. All we can glean from this study is that people who want to avoid high TMAO levels should not take L-carnitine supplements with their steak... and that mutant mice develop atherosclerosis when they are fed unnaturally high quantities of a form of carnitine not found in meat.

This study has led to additional research since, but as the science currently stands, the “red meat leads to high TMAO leads to high cholesterol which leads to heart disease” notion remains an unsubstantiated (and rather far-fetched) hypothesis that nevertheless continues to be

circulated as a reason to eliminate red meat from our diets.

WHO SAYS MEAT CAUSES CANCER

In 2015, the World Health Organization (WHO) issued a two-page report titled “The Carcinogenicity of Consumption of Red and Processed Meat,” warning that processed meat causes colorectal cancer in humans, and that red meat “probably” causes colorectal cancer in humans.²⁹ This document made headlines worldwide and continues to have a major impact on how people think about meat and health, but there are good reasons to question its findings.

The Epidemiological “Evidence” Against Meat

The vast majority of the studies used to prepare the report were nutritional epidemiology studies. The WHO looked at more than 800 epidemiological studies of red and processed meat and cancers of all kinds, but ultimately chose to focus only on colorectal cancer and put forth fifty-six human studies as “informative” on this topic.

Of the twenty-nine studies of unprocessed meat, fourteen suggested an association with higher risk for colorectal cancer, and fifteen did not. As we’ve learned, for epidemiological studies to suggest a cause-and-effect relationship between things like meat and cancer, their findings must be *consistent*. Since half of these studies pointed in one direction and half in the other direction, there is no reason to suspect that there is any connection between unprocessed meat and cancer worth exploring further. Of the twenty-seven studies of processed meat, eighteen suggested that processed meat was associated with a higher risk for colorectal cancer in humans and nine did not; while these findings are less conflicted, even if valid, they can only suggest a hypothesis about unprocessed meat that then must be tested in clinical experiments.

The Experimental Evidence Against Meat

The WHO cited only six experimental studies in their report (despite many more being available). Three were rat studies, two were human studies, and one was a rat-human study (a study of rats and humans, not of hybrid rat-

human creatures).

Red and Processed Meat in Rats

In these three studies, rats were first injected with powerful carcinogenic chemicals (azoxymethane or dimethylhydrazine) to hasten the cancer development process. Yes, you read that correctly. Next, they were fed various high-meat diets for one hundred days (roughly equivalent to ten human years). Some experimental diets contained adequate calcium and others were intentionally depleted of calcium (calcium protects cells against heme iron, which researchers believed to be the cancer-promoting ingredient within red meat). Lastly, they examined colon biopsies for potentially pre-cancerous changes. Only the calcium-deficient diets led to potentially pre-cancerous changes in rat colons, and even in these cases, no rat developed cancer.

The authors of one of these studies begin their paper with this striking statement: “In puzzling contrast with epidemiological studies, experimental studies do not support the hypothesis that red meat increases colorectal cancer risk. Among the 12 rodent studies reported in the literature, none demonstrated a specific promotional effect of red meat.”³⁰ Translation: they found a dozen rodent experiments testing the relationship between red meat and colon cancer and none of them had turned up any evidence against red meat. Surprisingly, not a single one of these twelve “red meat is fine” rodent studies were included in the WHO report. Selectively choosing to include studies that support one’s arguments while excluding those that don’t is known as *cherry-picking*.

Processed Meat in Humans

In the only study of processed meat in human subjects,³¹ researchers fed seventeen healthy men a low-antioxidant, low-calcium diet that included 6.3 ounces of poorly packaged ham for four days (ham was left unwrapped in the refrigerator for four days to allow exposure to air, which causes oxidative damage to the product). Then, rather than subject volunteers to colon biopsies, they tested their urine and stool for five different colon cancer *biomarkers*—substances that may be linked to colon cancer risk. Three of these biomarkers remained unaffected, including the two that are

considered the most reliable indicators of colon cell damage, but two increased: ATNC (apparent total n-nitroso compounds) and TBARS (thiobarbituric acid reactive substances). The relevance of both of these biomarkers to colon cancer has since been called into question by scientists,³² but nevertheless, when calcium and vitamin E were added back into the diet, these biomarkers did not rise in response to the poorly packaged ham.

Translation: Eating badly packaged ham for four days in a row probably won't increase your risk for colon cancer. If your diet contains adequate calcium and vitamin E, eating badly packaged ham definitely won't increase your risk for cancer.

Unprocessed Red Meat in Humans

The WHO report cited only two studies of unprocessed red meat in humans. Both were randomized controlled trials, but they were small, brief, and poorly designed.

In the first study,³³ researchers asked twenty-three volunteers to eat a low-fiber diet containing red meat for four weeks, and then to continue on that same diet supplemented with fiber for an additional four weeks. These unfortunate individuals then underwent rectal biopsies to look for a particular type of colon cell mutation called O6-MeG. Following the low-fiber phase, researchers did find more of these mutations, but when participants switched to the fiber-supplemented phase, this rise in mutations was completely blocked. It was later determined that O6-MeG is a poor biomarker for colon cancer risk, because this type of mutation naturally occurs so frequently in cells throughout our bodies that we produce an enzyme whose sole responsibility is to repair it wherever and whenever it occurs.³⁴

In the second study,³⁵ twenty-five volunteers were confined to a metabolic ward and fed one of three diets for ten days each: a vegetarian diet, a low-fiber diet containing red meat, and a high-fiber diet containing red meat. Following these interventions, researchers measured ATNC levels (the same biomarker used in the processed meat study above) and looked for a different colon cell mutation called O6-CMG. Once again, the low-fiber, red meat diet appeared worse, resulting in higher ATNC levels and

higher numbers of O6-CMG mutations than the vegetarian diet. However, the low-fiber meat group was fed refined carbohydrates in place of high-fiber foods, and if any volunteer in any group started losing weight, they were fed buttered marmalade bread to restore their original weight. We are not told which volunteers received these additional treats nor how often. Therefore, we don't know whether it was the presence of red meat, the presence of refined carbohydrates, and/or the lack of fiber that made the diet containing red meat appear worse than the vegetarian diet. Furthermore, it has since been discovered that our enzymes can also repair O6-CMG mutations, so these mutations may also be unreliable biomarkers for colon cancer risk.^{[36](#)}

There's a recurring theme here: researchers attempting to tie red meat to health problems change so many dietary variables that they hopelessly obscure the effects of red meat itself. Yet, when their overly complicated meat diets perform worse in their studies compared to meatless diets, they tend nevertheless to conclude that red meat must have been the culprit. It is remarkable the lengths some scientists go to in an attempt to demonstrate that red meat is unhealthy. Why not simply compare a diet containing red meat to a diet that is *otherwise identical* but that replaces red meat with a comparable quantity of tofu, white meat chicken, or fish?

Given that at least 768 of the 800+ epidemiological studies were either not worth considering or found no association between meat and cancer, that the majority of existing rodent studies found red meat did not promote colon cancer, and that none of the six experimental studies cited was designed in a way that can tell us whether red meat increases our risk for colon cancer, perhaps a more responsible and accurate title for the WHO report would have been "Vast Majority of Studies Find No Relationship Between Red Meat and Cancer."

In November 2013, twenty-three cancer experts from eight countries gathered in Norway to examine the science related to colon cancer and red and processed meat. They concluded: "The interactions between meat, gut and health outcomes such as CRC [colorectal cancer] are very complex and are not clearly pointing in one direction.... Epidemiological and mechanistic data on associations between red and processed meat intake and CRC are inconsistent and underlying mechanisms are unclear."^{[37](#)}

Translation: We don't know if meat causes colorectal cancer.

In 2022, a review of the nutrition epidemiology studies concerning unprocessed red meat and cancer found that the associations were too “weak and insufficient” to make conclusive recommendations about unprocessed red meat.³⁸

MISCELLANEOUS MEATY MYTHS

Defending claims against red meat is like playing Whac-a-Mole; as soon as one myth is bludgeoned back into its hole, another one pops up. Let’s briefly review some of the more common ones.

Seeing Red: Heme Iron Causes Cancer

In addition to its indispensable roles in animal biology, heme iron is what gives red meat its characteristic color and flavor. This is why some plant-based “meat” manufacturers spent seventy-five million dollars mass-producing genetically modified *leghemoglobin*—a form of hemoglobin located in the roots of soy plants—and inserting it into their patties.³⁹

So, could heme iron be the cancer-causing culprit lurking within red meat... and within bogus burgers? Not according to this 2015 statement written by David Klurfeld, PhD, the USDA’s National Program Leader for Human Nutrition, and one of the authors of the WHO report: “There is no data that normal levels of heme in human intestine contributes to any harm.”⁴⁰

Charred Meats Cause Cancer

Charring or wood-smoking meat produces polycyclic aromatic hydrocarbons (PAHs) and heterocyclic amines (HCAs), both of which have been shown to cause cancer in lab animals. The doses of these compounds that cause cancer in animals are 1,000 to 100,000 times higher than doses found in human food.⁴¹ Studies in humans are limited to epidemiological studies, and even these have been inconclusive.⁴²

HCAs can form not just in red meat but in any protein-rich food including fish and poultry. PAHs form in all plant and animal matter that has been exposed to high heat processing such as smoking, toasting, and frying. They are found in a wide variety of foods, including grilled

vegetables, cocoa products, and milk powder, but are extremely high in flour, cereals, and breads, which can contain levels more than a thousand times higher than in cooked meat.⁴³

Nitrates and Nitrites in Processed Meats Cause Cancer

Nitrates and nitrites are used in the production of processed meats like bacon, salami, and ham. However, they are also found naturally in many plant foods, often in very high amounts.⁴⁴ Nitrates and nitrites themselves have not been shown to cause cancer; however, they can react with other protein fragments to form *nitrosamines*,⁴⁵ chemicals that can cause cancer in laboratory animals. The majority of the nitrates in the typical Western diet come from vegetables, so if nitrates and nitrites do increase cancer risk, then not only should we limit our intake of processed meats, but we should also limit our intake of high-nitrate vegetables such as spinach and celery as well. Ounce for ounce, spinach contains more than eighty times more nitrate than hot dogs do, and celery powder is so high in nitrates that manufacturers use it to create processed meats, often proudly advertising that their products have “no nitrates or nitrites added except for those naturally occurring in celery powder.”

Meat Causes Constipation

Meat is the easiest food to digest. Stomach acid, intestinal enzymes, and bile efficiently dismantle its proteins into individual amino acids and its fats into fatty acids, and absorption into the bloodstream is virtually complete, with little to no waste left over to eliminate.⁴⁶ If you’re skeptical, simply ask yourself: 1) Have you ever seen pieces of undigested meat or fat leaving your body? 2) Have you ever seen pieces of undigested plant foods such as broccoli, nuts, seeds, peas, or corn leaving your body?

Meat Causes Obesity

Obesity is a relatively new epidemic, whereas meat is an ancient food. High insulin levels turn fat storage on, and low insulin levels turn fat storage off. Any way of eating that causes insulin levels to run too high too often can lead to unwanted fat gain, and the most powerful stimulators of insulin

release are foods high in refined carbohydrate ingredients such as sugar and flour. In a classic 1997 study measuring the insulin response to thirty-eight foods, beef's insulin index was 51, the insulin index of bananas was 82, white bread's insulin index was 100, strawberry yogurt's was 115, and jelly beans topped out at 160.^{[47](#)}

Meat Causes Diabetes

Type 2 diabetes is a disease characterized by persistently high blood glucose levels. Meat is incapable of causing unhealthy elevations in blood glucose because it is extremely low in carbohydrate. In the same 1997 study cited above, the glucose response to beef was by far the lowest of all thirty-eight foods tested. It's when you fry it in batter, douse it in sugary barbecue sauce, or tuck it into a sandwich that you may run into problems.

Meat Causes Hypertension

A 2020 meta-analysis of thirty-six RCTs found no effect of red meat on blood pressure in comparison to other protein sources.^{[48](#)} A recent study by Dr. David Unwin and associates found that patients prescribed a low-carbohydrate diet (that allowed meat) experienced significant reductions in blood pressure and reduced need for blood pressure medications.^{[49](#)} Since high insulin levels raise blood pressure and cause the kidneys to retain more sodium, lowering insulin levels by lowering carbohydrate intake could explain these benefits.

Meat Causes Kidney Disease

The number one risk factor for kidney failure is hyperglycemia and the number two risk factor is hypertension.^{[50](#)} Healthy kidneys can safely handle diets very high in protein; what they are NOT designed to handle is high glucose levels and high blood pressure. A 1930 study of two men who ate a 100-percent-meat diet for a full year revealed no signs of kidney problems whatsoever^{[51](#)} and modern studies of bodybuilders eating between 2.2 and 3 grams of protein per kg body weight per day (about three times the recommended amount) for months at a time found kidney function remained normal.^{[52](#)}

Meat Causes Gout

Gout is a type of arthritis often accompanied by high blood levels of a metabolic waste product called *uric acid*, which can crystallize in the joints and cause pain. Once called “the ailment of kings” because it mainly afflicted those who could afford to eat rich diets, gout now affects more than forty million commoners worldwide.⁵³ For centuries, it was believed that gout was caused by high meat intake, but there is no experimental evidence of this. It has recently become clear that, like so many other chronic diseases, gout is rooted in insulin resistance, which impairs the body’s ability to eliminate uric acid.⁵⁴

Meat Causes Inflammation

Molecules within red meat accused of causing inflammation range from saturated fat to polyunsaturated fat (arachidonic acid, an omega-6 found only in animal foods) to heme iron to neu5GC, a signaling molecule found only in mammal meat. However, a 2020 meta-analysis of twenty-four human RCTs concluded that red meat has no effect on blood markers of inflammation⁵⁵ whereas it is well established that hyperglycemia leads to inflammation throughout the body.⁵⁶

What If Eating Meat Breaks Your Heart?

The hypothesis that meat is dangerous to human health has driven a long and passionate hunt for evidence against meat that has thus far come up empty-handed. Rather than acknowledge that the trail has gone cold, this hypothesis continues not only to survive but to thrive—suggesting that it is far more likely to be rooted in feelings than in facts. As a psychiatrist, I have a healthy respect for feelings and understand that we humans make some of our most important decisions with our hearts rather than our minds. However, I also think everyone deserves to know the facts so that they can weigh them against their feelings and make their own decisions based on what matters most to them.

REAL PROBLEMS WITH MEAT

I am at peace with the biological fact that all life must consume life, but the way we treat the animals we depend on for food matters. Industrialized mass meat production systems create very real and deeply disturbing problems for the health and welfare of animals, humans, and the environment.

Factory-Farmed Fats

Just as is true for humans, when animals spend time in nature and eat a species-appropriate diet, their brains and bodies thrive. Whether factory-farmed, humanely raised, or wild, animal foods remain the safest and most nutritious foods we can eat, but that doesn't mean these animal foods are equally healthy, and one important difference would seem to be the quality of their fat. When an animal such as a hen, which is supposed to eat a diet of grass, wild plants, insects, and other small creatures is forced to eat a vegetarian diet based on corn, soy, and synthetic supplements, her body composition will change. She'll still be able to turn the starch into the glucose she needs, and she'll still be able to turn the proteins into the amino acids she needs (although she'll need a little help from amino acid supplements to provide those lacking in a vegetarian diet), but what will she do with all of the extra linoleic acid?

A hen's natural diet would provide omega-3 PUFAs (EPA and DHA) from grass, and plenty of saturated and monounsaturated fats from critters, but very little linoleic acid, whereas approximately 50 percent of the fat in corn and soy is linoleic acid.⁵⁷ Hens raised on corn and soy (or even worse, soybean oil, which is extremely high in linoleic acid) are forced to store this fragile fatty acid in their fat cells. The same is true of pigs, cows, and other livestock overfed on grains and legumes—and the same is true for us when we consume the meat (or eggs) of these animals. If you recall from [chapter 6](#), excess linoleic acid has a tendency to degrade into toxic byproducts with potentially negative health consequences.

Ethical and Environmental Concerns

Problems with factory farming of animals go well beyond how animals are fed to how they are housed, treated, medicated, and slaughtered. These serious concerns have led some to recommend that we stop eating animals

altogether, but it would be just as illogical to recommend that we stop eating plants altogether because pesticides and monocrop cultures endanger human health, destroy small animals and animal habitats, and damage topsoil. Industrialized plant and animal food production systems disrespect human employees, devalue consumer health, and pollute the land, water, and air. These are grave issues that we must face if we are to find a healthier, more sustainable way forward for plants, animals, and the environment we all share. It isn't the animals themselves that are damaging our health and our planet, it's the way we humans manage them. As dietitian founder of the Global Food Justice Alliance Diana Rodgers and paleo diet expert Robb Wolf write in their book *Sacred Cow: The Case for (Better) Meat*, "It's not the cow, it's the how."⁵⁸

Reviewing the science of the environmental impact of meat production and proposing solutions is beyond the scope of this book. However, my understanding is that if animals are raised well, they can be an indispensable part of the *solution* to many of our environmental and health challenges. The arguments in this arena are complex, nuanced, and well worth deeper exploration, so I refer you to the work of Nicolette Hahn Niman, the author of *Defending Beef: The Case for Sustainable Meat Production* and *Righteous Porkchop: Finding a Life and Good Food Beyond Factory Farms*. As an environmental lawyer and beef rancher who followed a vegetarian diet for much of her life, she is uniquely qualified both to prosecute the case against factory farming and defend the importance of thoughtfully managed animals to human and planetary well-being.

While it is always best to choose meats from wild animals or animals that have been humanely raised, allowed ample access to the outdoors, and fed a species-appropriate diet, these options are not always available, and can cost more. Explore local farms or farmers' markets where you can purchase meat as part of a community share, or order shipments of frozen animal foods from sustainable sources. Do the best you can, but don't let the perfect be the enemy of the good. When purchasing conventional animal foods in an ordinary grocery store, your best bet, according to Nicolette Hahn Niman, is beef:

Unlike other animals raised for meat, milk, or eggs, beef cattle start their lives with their mothers, nourished only by nursing and grazing. Most will continue to live in herds, on pasture or rangelands, for the first year of their lives. Regardless of how they are raised after that, they will never be confined to buildings or kept continually on concrete.... The more I saw and learned about the way dairy cows, pigs, chickens, turkeys, and egg-laying hens were raised, the more I regarded beef cattle as having the best lives of all animals in agriculture.^{[59](#)}

Calling for the end to production of these exceptionally nutritious, metabolically friendly whole foods is not the answer to our global mental health crisis, and in fact could easily make it worse. The challenge before us is to grow the foods we need in ways that minimize pollution and respect soil, ecosystems, and animals.

MEAT IS GOOD FOR YOU

- Meat (including red meat, seafood, and poultry) is a time-tested, evolutionarily appropriate whole food for human beings.
- There is no evidence that red meat (or any other type of meat) is harmful to any aspect of human health.
- Meat contains every nutrient we need, in its proper form, without antinutrients.
- Meat is good for gut health because it is non-irritating and very easy to digest.

How to Nourish, Protect, and Energize your Brain with Meat

- Choose healthy meats. Whenever possible, choose meats from wild animals or animals that have been raised humanely, allowed ample access to the outdoors, and fed a species-appropriate diet.
- Don't let the perfect be the enemy of the good. If you can't access or afford high quality meat, just do the best you can.

- It doesn't have to be red meat. It is very important to include some meat, seafood, poultry and/or eggs in your diet, but you do not need to eat the meat of mammals to meet your nutritional requirements. Shellfish, fatty fish, duck, and poultry liver are all examples of highly nutritious alternatives to red meat.
- Eat fresh. Choose unprocessed fresh (or freshly frozen) meats whenever possible.
- Don't fear natural animal fats. Fattier cuts of meat are more flavorful, more nutritious, and often less expensive. Unfortunately, pork and poultry fat from conventionally raised animals can be high in linoleic acid.
- Cook gently. Don't overcook meat, as this will damage nutrients and flavor. Trim away any burned or blackened areas of meats grilled or cooked at high temperatures.
- Don't overdo it. Overeating protein can promote higher insulin levels (and even slightly higher glucose levels in some people). Refer to [chapter 17](#) to estimate your daily protein requirements.

In my research, I have yet to find a credible, plausible health argument against including meat of any kind, red or otherwise, in the human diet. In fact, quite to the contrary; no other food group is nutritious enough, safe enough, or geographically accessible enough to recommend as the healthy foundation of the optimal human diet. If you could only afford to buy food from one food group, this would be the one to prioritize.

CHAPTER 11

Eggs and Dairy: Nature's Growth Formulas

Despite looking and tasting nothing alike, eggs and milk do have one important thing in common: they both exist to nourish new life. This special responsibility places them in a nutritional gray area; they are beloved, nutrient-rich, versatile, affordable sources of protein and other vital nutrients—making them particularly important foods for vegetarians and those who can't access meat—yet they also contain unique molecules engineered specifically to support immature animals of their respective species, and some of these molecules can be problematic for humans.

Since dairy products are high in saturated fat and eggs are high in cholesterol, thinking about these foods may conjure up images of coronary arteries slamming shut. The food industry capitalizes on fears of these whole foods by stripping eggs of their yolks, skimming fat off milk, and using what's left over to manufacture expensive processed products like Egg Beaters® and Go-gurt®. Worse still, it's becoming increasingly popular to completely replace these foods with plant-sourced products like Just Egg® and oat milk made from grain and legume protein extracts mixed with industrially refined vegetable oils. Manufacturers market these factory-made alternatives by boasting about what they *do not* contain (saturated fat, cholesterol, and animal protein), which distracts us from the problematic ingredients they *do* contain (industrially refined vegetable oils, low-quality plant protein extracts, and sugar).

In truth, eggs are a nearly perfect food, whereas dairy products—although they are nutritious—have complicated effects on human health and metabolism.

THE EGG: A MIRACLE OF NATURE

A box without hinges, key, or lid, yet golden treasure inside is hid.

—J. R. R. Tolkien, *The Hobbit*

The egg is an engineering marvel designed to house, defend, and nourish a bird embryo until it's ready to hatch. Whereas a human embryo receives a constant supply of nutrients and oxygen from its mother through the placenta, the bird embryo is physically disconnected from mother hen from day one and must obtain every nutrient required for healthy growth and development from the contents of its egg. During the three weeks that it takes for a chicken embryo to mature and hatch, its egg must be strong enough to withstand the weight of a brooding mother hen and protect the embryo from injury and infection, yet be porous enough to allow the developing bird to breathe.

The chick embryo feeds on the yolk, where the vast majority of an egg's vital nutrients reside. In fact, since egg yolks must provide all of the ingredients required to build an animal from scratch, they contain every nutrient we need (except for vitamin C, which the chick embryo makes for itself). Eggs are a particularly good source of choline (a fundamental component of cell membranes) and acetylcholine (a neurotransmitter central to learning and memory). If hens are raised on pasture, the fat of their yolks will contain more EPA and DHA—precious PUFAs that can be hard to find in many other foods—as well as more vitamin A, vitamin E, vitamin K1, vitamin K2, and vitamin D.¹

However, in comparison to meat, a few of the nutrients in eggs are harder for us to access.

The nutrient of most concern is iron. We absorb at most only about 3 percent of the iron in eggs, whereas we absorb between 10 and 20 percent of the iron in beef. This is partly because the form of iron found in eggs is non-heme iron,² and partly because a mineral-binding protein in the yolk called *phosvitin* interferes with our access to yolk's iron (and possibly its calcium and magnesium as well).³ Phosvitin survives cooking and is such a powerful iron magnet that each egg you consume reduces your ability to absorb non-heme iron from other foods in your digestive tract by 7 percent⁴ (it has no effect on heme iron).

Whereas egg yolk is plump with nutrients, egg white is a food desert.⁵ Egg white serves as a formidable barrier to bacteria, viruses, and fungi which may sneak into the egg through the thousands of microscopic air holes in its shell and try to infect the baby bird. Would-be invaders find this moat of slick, gelatinous proteins physically difficult to cross, extremely low in most nutrients, and teeming with antibacterial proteins. These defensive molecules include *protease inhibitors* that block bacteria's ability to digest egg proteins, *lysozyme*, which dissolves bacterial cell walls, and *avidin*, a powerful binder of biotin (vitamin B7).⁶ Fortunately, these antinutrients are largely destroyed by heat, so cooking egg whites increases their protein bioavailability from 65 percent to 95 percent and makes biotin easy to absorb.⁷

Eggs are also common culprits in food allergies and sensitivities, and the majority of the proteins that cause allergies are located in their whites.⁸ (We'll explore food allergies and sensitivities in [chapter 19](#).)

Nature's Most Perfect... and Most Dangerous Food?

Eggs are often described as nature's most perfect food, and they certainly do come close. Were it not for their low iron availability and lack of vitamin C, it would theoretically be possible to thrive on eggs alone. Eggs contain 6 grams of high-quality protein each, so they are an excellent choice for vegetarian diets. They are also extremely low in carbohydrate (less than one gram per egg), so they are metabolically safe for people with insulin resistance, and they contain about twice as much fat as protein, an excellent macronutrient ratio for ketogenic diets.

And yet, due to their cholesterol content, we are still advised to limit our egg intake, or to eat the least nutritious part of the egg (the white) and throw the most nutritious part (the yolk) away. Where does this advice come from?

Out of the Frying Pan...

Anti-cholesterol sentiments began stirring in the early 1960s, when the American Heart Association (AHA) announced that cholesterol in the foods we eat could build up in arteries and cause heart attacks.⁹ The science supporting this claim was far from settled. Nevertheless, under the

influence of the AHA, the committee responsible for writing the 1977 Dietary Goals for the United States recommended that Americans “reduce cholesterol consumption to about 300 mg per day;”¹⁰ this, despite acknowledging that there was “still controversy surrounding the exact relationship of dietary cholesterol to heart disease.”¹¹ The Dietary Goals evolved into the U.S. Dietary Guidelines, which (because eggs contain about 200 mg of cholesterol each) singled out the egg as a food to be eyed with suspicion, warning Americans in 1980: “moderate your use of eggs.”¹²

And so it was that for half a century, we were led to believe that the connection between eggs and heart disease was scientifically well-established and straightforward:

1. High blood cholesterol causes heart disease.
2. Egg yolks are rich in cholesterol.
3. Therefore, egg yolks cause heart disease.

Then, in 2013, the AHA, in conjunction with the American College of Cardiology (ACC), issued a new guideline abruptly reversing its position: “There is insufficient evidence to determine whether lowering dietary cholesterol reduces LDL-C”¹³ (LDL is *low density lipoprotein cholesterol*, aka “bad cholesterol”). Following suit, the U.S. Dietary Guidelines Advisory Committee (DGAC) reversed its position in 2015: “Available evidence shows no appreciable relationship between consumption of dietary cholesterol and serum cholesterol, consistent with the conclusions of the AHA/ACC report. Cholesterol is not a nutrient of concern for overconsumption.”¹⁴ Translation: eggs are innocent—eat as many as you like.

Why the about-face? Quite simply because decades of laboratory studies, animal studies, and human clinical trials could find no clear connection between how much cholesterol people ate, how high their cholesterol levels were, and whether they would develop heart disease. There were even plenty of human clinical trials demonstrating that eating eggs provided heart health *benefits*.¹⁵

... Into the Fire

You would think that these official proclamations from both the AHA and the DGAC would have been enough to declare eggs innocent once and for all, but in some circles, it seemed only to fan the flames of the debate as nutrition epidemiology studies from both sides of the debate have since been hurled back and forth, resulting in headline whiplash and public confusion (emphases mine):

- 2018 “An Egg a Day May **Reduce** Heart Disease Risk, Study Finds”¹⁶ (Peking University)
- 2019 “Eggs **Raise** the Risk for Heart Disease and Death”¹⁷ (Northwestern University)
- 2020 “Moderate Egg Consumption Gets the **Green Light**... Again”¹⁸ (Harvard University)
- 2021 “Eating Just Half an Egg with Yolk a Day Increases **DEATH** Risk 7%”¹⁹ (Zhejiang University)

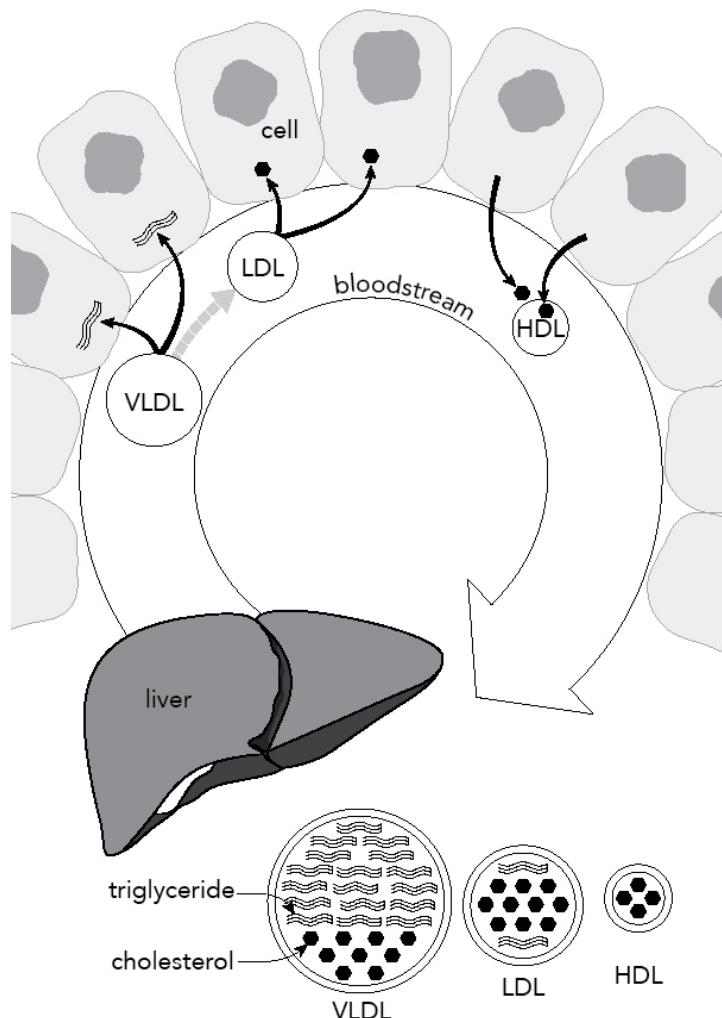
Yet notice that even the pro-egg headlines still advise us to be cautious about how many eggs we eat. For example, the Harvard study mentioned above, which was co-authored by Professor Walter Willett, concluded that “moderate egg consumption (up to one egg per day) is not associated with cardiovascular disease risk.”²⁰ This conclusion is only somewhat reassuring, as it invites us to worry that eating more than one egg per day might still be dangerous. However, a close reading of their paper reveals that eating *one or more* eggs per day was not associated with heart disease risk. The authors had inexplicably placed an arbitrary limit on egg consumption that was not supported by their own work.

Despite overwhelming evidence that dietary cholesterol in general and eggs in particular are completely safe for your blood cholesterol levels and your heart, the U.S. Dietary Guidelines continue to recommend that “dietary cholesterol consumption... be as low as possible without compromising the nutritional adequacy of the diet.”²¹ The result of these scrambled messages about eggs is that many people still believe eggs are risky, when the truth is, eggs are among the safest, most nutritious foods you can eat.

DEEPER DIVE: UNDERSTANDING CHOLESTEROL

We have been taught to think of LDL as “bad cholesterol,” but your body makes it for a very good reason. LDL itself isn’t actually cholesterol; it’s a pod made of protein and fat called a *lipoprotein particle* that carries fat and cholesterol molecules around in your bloodstream. When lipoproteins pick up cargo, they expand, and when they drop off cargo, they shrink, so their size and density are constantly changing. Since cholesterol is denser than fat, pods full of fat are lower in density than pods full of cholesterol.

LDL (low-density lipoprotein) pods begin their voyage as *very* low-density lipoprotein pods (VLDL) in the liver, which loads them up with lots of fat along with some cholesterol and ships them out into the bloodstream. (VLDL is also considered “bad” but standard cholesterol tests usually don’t measure it.) VLDL’s top priority is delivering energy-packed fat molecules to hungry cells throughout the body. As VLDL circulates and loses fat, it gradually becomes smaller and denser until most of what is left inside is cholesterol. At this point, it is called an LDL particle, and its priority becomes *cell maintenance*. Since cell membranes require cholesterol, wherever existing cells need repair or new cells need to be created, cholesterol will be in high demand, so LDL’s job is to supply cholesterol building blocks to construction sites around the body.



CHOLESTEROL AND FAT TRANSPORTATION SYSTEM

The liver sends very-low-density lipoproteins (VLDLs) carrying triglycerides and cholesterol into the bloodstream. As VLDL delivers triglycerides to cells, it gradually shrinks to become low-density lipoprotein (LDL), which distributes cholesterol to needy cells and then returns to the liver. High-density lipoprotein (HDL) collects damaged or unneeded cholesterol from cells and carries it to the liver to be recycled or removed from the body.

Suzanne Smith

It's common to view cholesterol as nothing more than an unwanted intruder that worms its way into blood vessel walls and causes heart attacks and strokes, so LDL is labeled "bad" because it delivers cholesterol to cells. High-density lipoprotein (HDL) particles are viewed as "good" because they *remove* damaged or discarded cholesterol molecules from cells and return them to the liver. Just as it doesn't make sense to think of saturated

fats as bad and unsaturated fats as good, it doesn't make sense to think of LDL (and VLDL) as bad and HDL as good. We need *all* of our lipoprotein particles for the distribution system to function. Without VLDL and LDL, cells can't receive shipments of cholesterol they need to maintain themselves, and they can't receive shipments of fat to burn for energy—and don't most people want to burn fat?

So, how did LDL become the black sheep of the lipoprotein family?

Cholesterol and Cardiovascular Disease: Guilt by Association

Since cholesterol is found inside the atherosclerotic plaques associated with heart attacks and strokes, it was assumed that LDL caused those plaques to form. However, decades of research have yet to produce credible support for this idea; in fact, there is ample evidence *against* it.²² We still don't fully understand how or why cholesterol finds its way inside blood vessel walls—but most experts now agree that LDL only gets trapped inside *unhealthy* blood vessels.

The first step in plaque formation isn't high LDL levels, it's damage to the *endothelium*—the layer of cells that lines the inside of blood vessels.²³ Many things can harm your endothelium, but the most important examples are smoking, high blood glucose levels, and high blood pressure. When damage occurs, a blood clot will form to try to protect the area, but if your clotting system is overactive or your immune system isn't working properly to heal the wound, the long, complicated process of plaque formation will begin, and eventually, cholesterol will make its way into the vessel wall—*regardless of what your blood LDL level happens to be*.

For example, a 2009 study of 136,905 people who had just been admitted to the hospital with heart attacks found that nearly half of them had LDL levels below 100 mg/dl, which was the healthy limit recommended by expert guidelines at that time.²⁴ This means that your LDL level is no better than a coin toss at predicting your risk of a heart attack.

The truth is that a simple LDL level cannot tell you whether or not you will have a future heart attack or stroke;²⁵ it has to be interpreted in the context of the bigger clinical picture. For instance, if your LDL is high, it could simply mean that you are vigorously burning fat for energy! This may

be why some individuals following a low-carbohydrate, high-fat diet see their LDL rise, and researchers are exploring this question.²⁶

Meanwhile, Back at the Breakfast Table...

How many eggs are safe to eat?

If you add three eggs per day to people's usual diets for weeks in a row, in most cases, their cholesterol level won't budge. About one-third of subjects will see a small rise in LDL but they will also see a small rise in HDL, so their overall cholesterol ratio (a commonly used estimate of overall cardiovascular risk) doesn't change.²⁷

Your body isn't a bucket that you pour cholesterol into until it spills over. Your digestive tract has sophisticated controls in place to regulate how much cholesterol you absorb from food, how much your cells manufacture, and how much you eliminate in your bile.

In one interesting case, an eighty-eight-year-old man who had consumed at least two dozen eggs every day for fifteen years maintained a total cholesterol level of 150 to 200 mg/dl.²⁸ Researchers found that his body only absorbed about 18 percent of the cholesterol he ate and had doubled its rate of cholesterol removal to keep his system in balance.

If you "overeat" cholesterol, your body will respond by changing its behavior, and the same is true if you "undereat" cholesterol. Nearly every cell in your brain and body is equipped to manufacture cholesterol, so even if you eat a cholesterol-free vegan diet, your body will see to it that there is always plenty of cholesterol in your blood. A study of more than 3,500 women found there was no significant difference between the LDL levels of vegans, vegetarians, and omnivores.²⁹ It's even possible for people eating vegan diets to have "high" cholesterol.³⁰

The amount of cholesterol in your diet doesn't determine the amount of cholesterol in your blood—your metabolism does.

SO, ABOUT THOSE EGGS

- Cooked (whole) eggs are good sources of every nutrient we need except for iron and vitamin C.
- Egg yolk is a good source of choline, vitamin B12, and vitamin A,

and is one of the few sources of MK-4, the brain's preferred form of vitamin K2.

- Egg yolks are rich in cholesterol, a vital nutrient. The number of eggs you eat per day has no bearing on your blood cholesterol level.

Tips for Nourishing, Protecting, and Energizing your Brain with Eggs:

- Eggs are very poor sources of iron, so if you don't eat meat, be sure to include other sources of iron in your diet.
- To maximize their nutritional benefits, cook eggs before eating and don't throw away the yolk—that's where the nutrients are.
- Eggs contain about twice as much fat as protein, an excellent macronutrient ratio for ketogenic diets.
- Egg allergies and sensitivities are not uncommon, so if you have unexplained physical or mental health symptoms, consider a thirty-day egg-free experiment to explore this possibility.

Cooked (whole) eggs feature a nearly perfect nutrient profile. Versatile, affordable, and available almost everywhere, eggs are among the healthiest foods you can eat. Unless you have an allergy or sensitivity to eggs, it is safe to enjoy them to your heart's content.

THE COMPLICATED NATURE OF DAIRY

Wild aurochs (prehistoric cattle) were first domesticated in the Middle East about 10,000 years ago,³¹ and the earliest archaeological evidence that humans were consuming milk on a regular basis dates back only about 6,000 years to southern England.³² Today, foods made from the milk of various mammals—mainly cows, buffalo, goats, sheep, and camels—are considered staple foods in most countries,³³ but are only rarely consumed in some parts of the world, such as China, Indonesia, and North Korea.³⁴

I have found the experimental elimination of dairy to be a very useful tool in my clinical practice, particularly for addressing binge eating, digestive issues, and unexplained pain syndromes. This may be partly due

to the complexity of the molecules dairy products contain, and/or the fact that human physiology hasn't had much time to adapt to this food group, given how recently it was added to the Western menu. Research into the potential connection between dairy and mental health problems is thin, but let's review the compelling scientific information that is available, with the hope that it might inspire you to explore how this complicated food affects you.

MILK MICRONUTRIENTS

Like the egg, milk is often called one of nature's most perfect foods, but its nutritional profile isn't quite as strong. Milk is a famously good source of calcium, but contains no vitamin D, so several countries (the United States, Canada, Norway, Sweden, and Finland) fortify milk with vitamin D because we can't absorb calcium without it.³⁵ Milk is also naturally low in iodine, but most milk producers in industrialized countries add iodine to dairy cattle feed and use an iodine solution to disinfect udders prior to milking in a practice known as "teat dipping."³⁶ Cow's milk is also very low in iron, even compared to many other mammal milks, perhaps because industrial dairy practices push them to produce such large quantities of milk.³⁷ Milk is a very poor source of the omega-3 fatty acids EPA and DHA, regardless of how the cow was raised. For example, most health organizations recommend adults obtain about 250 mg per day of EPA and DHA. Although milk from grass-fed cows does contain about two-thirds more EPA than milk from grain-fed cows, an eight-ounce glass still only provides about 10 mg of EPA (and only about 2.5 mg of DHA).³⁸ Milk contains no vitamin C, because calves rely on vitamin C stores built up in utero until they reach about four months of age when they become capable of making their own.³⁹

MILK MACRONUTRIENTS

Milk is the only animal food that contains substantial quantities of all three major macronutrients: protein, fat, and carbohydrate. *This is nature's recipe for rapid growth.* Many of milk's macronutrient molecules are uniquely required by immature mammals, so you won't find them in meat. The

quantity and type of macronutrients also varies from one species to another, as different mammals have different needs.

Carbohydrate (Lactose)

The only type of carbohydrate that milk contains is *lactose*, a special sugar found only in milk. Each lactose molecule consists of a glucose molecule bonded to another simple sugar molecule called *galactose*. When we are babies, our intestines produce an enzyme called *lactase* that breaks the bond between these two simple sugar molecules, allowing us to absorb glucose and galactose into the bloodstream. All other mammals permanently lose the ability to produce this enzyme after weaning because they no longer need it, and before the taming of the aurochs, early humans were no different—they lost their ability to digest lactose in early childhood as well.⁴⁰ After the Agricultural Revolution, certain groups who relied more significantly on dairy farming—mostly in northwest Europe—developed a genetic mutation that allowed some of them to produce lactase into their adult years; this trait is called *lactase persistence*. However, the majority of us—nearly 70 percent—still cannot properly digest lactose.⁴¹

Region/Ethnicity*	Prevalence of Lactose Malabsorption ⁴²
Scandinavia	3–5%
Great Britain	5–15%
Germany	15%
Austria	15–20%
North American whites	15%
Finland	17%
France	17% (northern); 65% (southern)
Italy	20–70%
India	30% (northern); 70% (southern)
North American Hispanics	53%

The Balkans	55%
South America	65–75%
North American blacks	80%
Africa	70–90% (exceptions: Bedouins, 25%; Tuareg, 13%; Fulani, 22%)
Central Asia	80%
Eastern Asia	90–100%

*Terms in this column were those used in the original study; regions/ethnicities not included here were not represented in the study.

Without sufficient lactase, any lactose you consume will cruise completely intact through your small intestine and all the way down into your colon, where it will encounter swarms of gut bacteria. However, they don't possess lactase either, so they can't digest it down into glucose and galactose for us. Instead, bacteria ferment (partially digest) the lactose, releasing fatty acids and gases in the process. As anyone with severe lactose intolerance knows, these gases can cause significant bloating, pain, and/or diarrhea.⁴³ Since lactose is identical across all species, if you are intolerant to the lactose in cow's milk, you will be intolerant to the lactose in all other milks as well. Fortunately, lactose intolerance is not a dangerous condition; it's simply a sign that your digestive system has matured and you no longer need milk.

The dairy products highest in lactose are milk, milk powder, condensed milk, buttermilk, cottage cheese, ice cream, yogurt, and kefir. Dairy products that contain mostly fat such as butter, ghee, and cream have very little lactose, and dairy products that have been aged for a long time, such as hard cheeses and ripened cheeses, are very low in lactose because the bacteria used in the fermentation process have already “eaten” the lactose for you. Here's a tip: If the nutrition label on a dairy product lists the carbohydrate content as zero mg, then the product contains little to no lactose. Lactose-reduced and lactose-free products are available in some areas, and lactase supplements can help ease symptoms.

However, if even low-lactose dairy products don't agree with you, you may have an intolerance to dairy protein.

Dairy Proteins

If left out too long, milk will eventually separate into solid lumps (curds) of calcium-rich proteins called *caseins* that sink to the bottom and a watery substance containing dissolved *whey proteins* that float to the top. Caseins and wheys are the two main categories of protein found in all types of milk, and they are designed not only to nourish the newborn, but also to transmit information from mother cow to calf that regulates genes, trains the immune system, builds the microbiome, and stimulates growth pathways.

Caseins Are Complicated. Eighty percent of the proteins in cow's milk are caseins (compared to less than 50 percent in human breast milk).⁴⁴ Casein molecules are sticky—so sticky, in fact, that they were historically used as an active ingredient in wood glue. This property causes caseins to clump together in the gut and form large clots of protein that take a long time to digest, essentially serving as an extended-release source of nutrients to sustain the calf between feedings. In the human gut, cow caseins can take so long to dismantle into individual amino acids that indigestion or constipation may occur.

Partially digested casein fragments may also cause problems of their own for some people. This appears to be particularly true in the case of *A1-beta casein*, a type of casein found in the milk of certain herds descended from northern European stock, including Holstein cows. In the Western world, Holsteins have long been bred with other dairy cattle, so most milk sold in Western markets now contains A1-beta casein. (This is in contrast to A2-casein, found in the milk of purebred Asian and African cattle breeds, as well as certain mainland European stock such as Jersey cows.) Incomplete digestion of A1-beta casein can generate *beta-casomorphins*—morphine-like molecules with narcotic and inflammatory properties.⁴⁵

If these casomorphins bind to naturally existing opiate receptors in the human gut, they may slow down digestive machinery and lead to constipation, just as morphine and other narcotic medications can. They can also trigger inflammation, which may irritate or damage the gut lining.⁴⁶ This is important, because inflammation in the body can also cause inflammation in the brain.⁴⁷

Scientific studies of dairy and brain inflammation are few and far between, but what we do have is intriguing. The most striking example:

There are well-documented cases of infants and children with epilepsy whose seizures completely resolved when dairy was eliminated from the diet, whether they tested positive for cow's milk allergy or not.⁴⁸ These experiences tell us that sensitivity to cow's milk can profoundly destabilize brain chemistry in susceptible individuals.

It is common for parents of children with autism spectrum conditions to experiment with diets free of casein and gluten in hopes of seeing improvement in autism symptoms, and some nonrandomized trials have reported significant benefits with this intervention. However, the few small randomized controlled trials that have been conducted so far have not found benefits.⁴⁹

Is Dairy Addictive? Most low-carbohydrate and ketogenic diets emphasize heavy cream, butter, and cheese because they are naturally low in carbohydrate, but quite a few of my patients feel "addicted" to dairy products, which can lead to cravings, binge eating, and unwanted fat gain. One reason for this may be that casein stimulates the release of a hormone called IGF-1 (insulin-like growth factor type I),⁵⁰ which stimulates growth pathways in our bodies⁵¹ and increases appetite.⁵² Casomorphins may also have something to do with it, because they can cross the blood-brain barrier and stimulate opiate receptors in the brain. However, the opiate-like effects of casomorphins are fairly weak compared to drugs like morphine, and (to make matters more complicated) some dairy proteins even have anti-opiate properties.⁵³ Perhaps the allure of dairy products is that processing methods—even traditional methods like fermentation, churning, and salting—concentrate their ingredients and intensify their flavor profiles. There isn't enough research to be certain about what drives some people to lose control over their appetite and eating behavior when eating dairy products, but if you struggle with overeating, a dairy-free experiment is well worth trying.

A woman in her sixties with a long history of overeating consulted with me because she was trying to transition to a ketogenic diet but couldn't make it past day three without intense cravings and urges to binge. The plan she was following allowed for up to four ounces of cheese per day, so she had been enjoying cream cheese as a daily snack. When I suggested she try setting all dairy aside as an experiment, she was easily able to make it past day three, get into ketosis, and enjoy excellent appetite control without making any other changes to her menu.

Whey Proteins. Whey proteins are popular with bodybuilders because they are rapidly digested into individual amino acids, stimulating an insulin surge that supports the growth and repair of muscle tissue after exercise. They are also popular with processed food companies who use them to manufacture “keto-friendly” products such as shakes, chips, cereals, and bars. Although whey protein contains no carbohydrate and doesn’t raise glucose levels, it raises insulin levels substantially, so it can turn fat-burning off and reduce ketone levels. In fact, the insulin response to whey is almost as dramatic as the insulin response to pure glucose.⁵⁴ (Buyer beware: just because the label says “keto-friendly” doesn’t necessarily mean the product is ketogenic or friendly.)

Dairy Fat

Dairy fat is interesting in that it is the only animal fat that contains more saturated fat than unsaturated fat. However, as we have already established, saturated fat in and of itself is not unhealthy. Furthermore, the fat within full-fat dairy products is where the “fat-soluble vitamins” reside. For example, grass-fed butter is a good source of vitamin A and one of the few sources of MK-4, the brain’s preferred form of vitamin K2.

Compared to lactose and dairy proteins, which are fairly common causes of dairy intolerance, the fat within dairy products may actually be their least problematic ingredient. Some of my patients with dairy intolerance seem to do better with butter (which is mostly dairy fat), or better yet, ghee (which has been heated to remove as much protein as possible). However, some of my patients do poorly with all dairy products, including ghee, suggesting either that they are sensitive to trace amounts of protein that may remain, or that they are reacting to some unknown problematic element within the dairy fat itself. Unfortunately, there is very little research on the topic of dairy fat intolerance, so we can only speculate.

Dairy and Insulin Resistance

Perhaps the most compelling reason to reconsider the role of dairy in your diet is that a number of human clinical studies have found that dairy products raise insulin levels and cause insulin resistance. In one particularly interesting experiment,⁵⁵ researchers in Denmark asked twenty-four healthy

eight-year-old boys to add 53 grams of animal protein per day to their usual diets—half of them were to consume this additional protein in the form of 8.8 ounces of lean meat and the other half were to add two liters of skim milk. After seven days, the fasting insulin levels and insulin resistance of boys in the meat group remained unchanged, whereas the fasting insulin levels of those in the milk group had *doubled*, and insulin resistance had increased by 75 percent.

DAIRY, CALCIUM, AND BONE HEALTH

We're told the biggest reason we need dairy is because it is rich in calcium, which supports bone growth and prevents osteoporosis. And yet, our fellow mammals all appear capable of generating complete sets of bones and teeth without consuming milk beyond early life. Since there is no reliable way to diagnose calcium deficiency, it is hard to know how much calcium we really need and whether we are getting enough from the foods we eat, but some research suggests that dairy products may not be essential to bone health. For example, countries where milk intake is high, such as Norway, Sweden, and Denmark, report the highest rates of hip fracture in the world, whereas China, Indonesia, and India, where milk intake is low, report the lowest rates of hip fracture.⁵⁶ Also, osteoporosis was rare prior to the industrial age and occurs much more often in industrialized areas of the world,⁵⁷ suggesting that it may be related to aspects of our modern lifestyle other than how much milk we consume.

Bones do need calcium, but total bone health is about much more than the amount of calcium in your diet. Optimal absorption of calcium requires vitamin D3, and optimal binding of calcium to bone requires vitamin K2. As mentioned earlier, keeping glucose and insulin levels in a healthy range helps to minimize inflammation, which can damage bone over time. Sodium and caffeine both increase the amount of calcium your body loses in the urine. Bone is made largely of

protein, so obtaining enough protein to support bone structure and engaging in weight-bearing exercise to stimulate bone metabolism is essential.⁵⁸

DOES DAIRY REALLY DO A BODY GOOD?

- Dairy products are excellent sources of calcium and good sources of most other essential nutrients except for vitamin C, vitamin D, iron, EPA, and DHA (and sometimes iodine).
- Dairy products contain high quality protein that provides all essential amino acids.
- Lactose intolerance is exceedingly common (and evolutionarily appropriate).
- Allergies and intolerances to dairy proteins are common, and may contribute to gastrointestinal distress, acne, eczema, asthma, migraine, chronic pain syndromes, and other inflammatory conditions.
- Dairy products contain unique molecules that can contribute to insulin resistance, metabolic dysfunction, appetite dysregulation, and weight gain.
- All dairy caseins are difficult to digest. Milk from certain breeds of cattle contain A1-beta casein, which may also have narcotic and inflammatory properties.

Tips for Nourishing, Protecting, and Energizing Your Brain with Dairy

- If you have never tried a dairy-free experiment, I encourage you to set aside all dairy products for thirty days to see how you feel.
- If you find dairy products to be addictive, inflammatory, constipating, or fattening—or if they make you feel depressed, unfocused, or fatigued—it would be wise to eliminate them from your diet permanently.
- If you decide to go dairy-free and you’re worried about calcium,

know that calcium is present in certain other animal foods, including shrimp and small, soft-boned fish such as sardines, anchovies, and mackerel. Some plant foods also contain calcium, with the most bioavailable sources being cruciferous vegetables.

- If you feel better without dairy but don't want to permanently avoid it, try reintroducing different dairy products one at a time to see which ones bother you least. For example, if dairy protein is your issue, you may tolerate low-protein dairy products such as ghee and butter fairly well.
- You might try seeking out "A2" milk so you can compare its effects to A1 milk. Pure A2 milk is usually clearly labeled as such to attract consumers interested in its potential health benefits.
- If you suspect you are sensitive to lactose, look for dairy products with a carbohydrate count of 0 grams per serving. You may find fermented dairy products such as hard cheeses, sour cream, and full-fat yogurt to be gentler on your system because fermentation removes lactose and partially breaks down dairy proteins. (However, fermentation also changes dairy proteins in ways that can make some people feel worse; see [chapter 19](#).)
- If you choose to include dairy in your diet, full-fat dairy products are more nutritious, more satisfying, and less processed.

Milk is a species-specific growth formula and therefore the most complex food on Earth—and that's even before we churn, ferment, and skim it into the staggering variety of dairy products available today. We know so little about how each of these products affects our mental and physical health, but what we do know strongly suggests that the human diet should not include milk beyond early childhood—particularly not the milk of other species. Be careful with this food group. Even if you can't pinpoint any negative effects, if you are pursuing optimal mental and physical health, it may still be wise to avoid dairy entirely.

CHAPTER 12

Grains, Beans, Nuts, and Seeds: Consumer Beware

Seeds are our most durable and concentrated foods. They're rugged lifeboats, designed to carry a plant's offspring to the shore of an uncertain future.

—Harold McGee, *On Food and Cooking*

We think of grains, beans, nuts, and seeds as unrelated, but crack any of them open and you'll find a tiny embryo crouched inside. Even though we only use the word "seed" for one branch of this family, deep down, *all of these foods are seeds*.

- Grains are the seeds of grasses (wheat, corn, rice, oats, etc.)
- Beans are the seeds of legumes (peas, soybeans, lentils, etc.)
- Nuts are the seeds of trees (almonds, walnuts, pistachios, etc.)
- "Seeds" are (mostly) the seeds of flowers (sunflower seeds, poppy seeds, sesame seeds, etc.)

To avoid confusion, we'll use "seed foods" as an umbrella term to refer to all grains, beans, nuts, and seeds.

Just as dairy products are the most complicated animal foods on Earth, seed foods are the most complicated plant foods on Earth. Protecting and nurturing the future generation of one's species is no easy task, and these weighty responsibilities are what make these simultaneously the most nutritious and most problematic plant foods you can eat. Avoiding the seed food family as much as you can—especially grains and legumes—is one of

the most important things you can do to protect your brain and overall health. However, if you want or need to include this food group in your diet, I will show you how to make safer, healthier choices.

THE BENEFITS AND RISKS OF SEED FOODS

Grains, beans, nuts, and seeds are the most nutritious parts of plants because they must contain every nutrient an embryo needs to develop the roots, shoots, and leaves that will ultimately establish the seedling's independence. All seed foods contain protein to provide the amino-acid building blocks their seedlings need to grow, but beans and nuts are generally richer in protein than grains and seeds, making foods like soybeans, peas, and nuts critical staples in vegan diets and in the diets of great numbers of people around the world with limited access to animal protein. Despite being much lower in protein and fat than other seed foods, grains are less expensive and easier to grow in large quantities than beans, nuts, and seeds, so they are an important source of carbohydrate calories worldwide and figure prominently in the dietary guidelines of most countries.

The nutritional advantages of grains, beans, nuts, and seeds must be weighed against the fact that every member of the seed food family must not only nourish its precious cargo, but also defend it from the elements and from predators large and small until conditions are right to sprout. Animals protect themselves from being eaten using action-oriented strategies like growling, biting, charging, and fleeing, but plants are stationary and voiceless. These physical limitations can make them appear helpless and vulnerable—but looks can be deceiving. In fact, seed foods are the most heavily armed of all plant foods. Not only do they wear tough exterior shells and hulls, but concealed within these rugged structures are invisible stockpiles of chemical weapons: toxins and antinutrients that pose risks to human health.

THE BIRTH OF AGRICULTURE

From deep within Israel's Dead Sea rift, archaeologists have unearthed remnants of almonds, pistachios, and other nuts along with pitted stones

that were likely used as nutcrackers, suggesting that our prehistoric ancestors were consuming nuts as long ago as 780,000 B.C.¹ By comparison, although stone tools were used in Mozambique to grind sorghum grains about 100,000 years ago, it wasn't until about 11,000 years ago that humans appear to have begun consuming grain in significant quantities on a regular basis, as evidenced by the discovery of large storage structures containing barley and oats in the West Bank.² As for legumes, evidence of domesticated fava beans in Israel³ and lentils in Southwest Asia⁴ dates back as far as 10,000 years ago.

Most experts trace the birth of agriculture to the Fertile Crescent about 11,000 years ago. Arching from the Persian Gulf to Northern Egypt, this “cradle of civilization” was blessed with rich soils irrigated by the rhythmic flooding of the Tigris and Euphrates rivers. Over the next 5,000 years, farming would become a way of life almost everywhere, but it appears we paid a price for it, both socially and biologically. As historian Yuval Noah writes in his book *Sapiens*, “Cultivating wheat provided much more food per unit of territory, and thereby enabled *Homo sapiens* to multiply exponentially... This is the essence of the Agricultural Revolution: the ability to keep more people alive under worse conditions.”⁵ Anthropological records reveal that individuals living in agricultural societies were generally of shorter stature than their hunter-gatherer relatives who came before them, and that their bodies showed evidence of mineral deficiencies, malnutrition, and infectious diseases.⁶ Thousands of years may seem like enough time for us to adapt to eating these foods, but from an evolutionary standpoint it is the blink of an eye. Our species, *Homo sapiens*, is about 200,000 years old, so we have only been consuming grains and legumes for 5 percent of our history, at most.

Seed Macronutrients

Like milk, all seed foods contain nature's recipe for growth—a combination of all three macronutrients in fair amounts: protein, fat, and carbohydrate. However, while cow's milk macronutrients are intended for the young of another mammalian species, seed macronutrients are intended for the young of a completely different *kingdom*: the plant kingdom (as opposed to the animal kingdom, to which we belong). It is therefore not surprising that

their macronutrient composition is less than optimal for human nourishment. We are often told that these foods are good for us because they are rich in plant protein and complex carbohydrates, low in saturated fat, and cholesterol-free. All true statements, but the *whole truth* is that their proteins usually don't contain the balance of amino acids we need, most of their proteins and carbohydrates are difficult to digest, and there is no reason to avoid saturated fat or cholesterol.

Seed Food Proteins. It's certainly possible to obtain all of your essential amino acids from seed foods alone, but it requires special knowledge, extra effort, and (almost always) more food.

All seed foods do contain all nine essential amino acids, but usually not in quantities sufficient to meet our needs. For example, grains tend to be low in lysine, and legumes tend to be low in methionine and cysteine.⁷ Seed proteins are also guarded by antinutrients that make their proteins less available to us. For example, many seed foods contain protease inhibitors that interfere with the enzymes in our gut that break proteins down into individual amino acids. Cooking inactivates most protease inhibitors,⁸ but there are still other anti-protein factors in seed foods to contend with, including phytate (which heat can't completely neutralize) and tannins (which are indestructible).

To help navigate this seed protein obstacle course, scientists have developed a protein quality index called the *digestible indispensable amino acid score* (DIAAS). This scoring system takes the digestibility and the amino acid balance of various proteins and rolls them into a single number. The table below lists the DIAA scores of selected plant and animal foods.

Protein Source	DIAAS ⁹	Quality
Whole milk	114	High quality
Egg (hard boiled)	113	
Beef	112	
Chicken	108	
Tilapia	100	
Tofu	97	Good quality

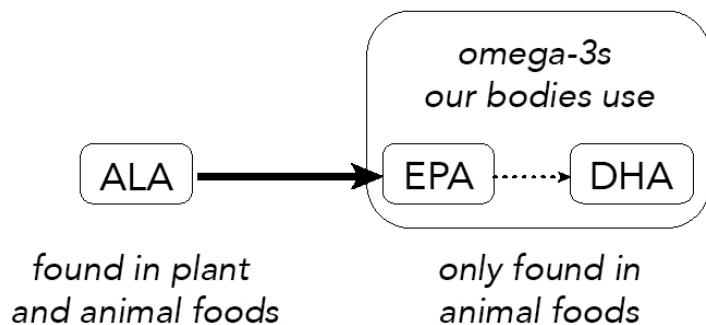
Chickpea	83	
Pea protein concentrate	82	
Cooked rice	59	Poor quality
Cooked peas	58	
Almonds	40	
Seitan (wheat)	28	

A DIAAS of 100 or above means that you can meet or exceed your daily requirement for all essential amino acids by eating that food alone. Notice that all animal foods have DIAA scores of 100 or higher, indicating that they contain highly digestible forms of every amino acid we need. Scores for tofu, chickpeas, and pea protein concentrate fall between 75 and 100, and are therefore considered good quality proteins. The remaining plant proteins are considered poor choices, as they all score well below 75. With the notable exception of tofu, if you are trying to meet your amino acid requirements using plant foods alone, you will need to mix and match your protein choices properly to avoid deficiencies over time.

Seed Food Fats. Nuts and seeds tend to be much higher in fat than grains and legumes. Except for coconuts and palm kernels, which are both very high in saturated fat, most of the fats in nuts and seeds are MUFAs (mainly oleic acid), and PUFAs, two of which we are told are essential: linoleic acid and the omega-3 alpha-linolenic acid, or ALA.

As discussed in [chapter 6](#), linoleic acid is easy to find in a wide variety of plant and animal foods, but ALA is much harder to come by. Many animal foods contain some ALA, but there are a few plant foods such as flaxseeds, walnuts, and chia seeds that are rich in ALA, so they are prized (and marketed) as excellent sources of omega-3s—but *ALA is not the omega-3 we should be looking for*. The omega-3s we need are EPA (which helps your immune system fight inflammation) and DHA (critical for brain development, brain cell signaling, brain immune system function, and energy production). In fact, the *only* thing we need ALA for is to turn it into EPA and DHA.¹⁰ Building DHA molecules for the brain would be a fine use of ALA, but unfortunately our capacity to convert ALA into DHA is

extremely low; nonpregnant women convert at most about 9 percent of the ALA they consume into DHA, and the conversion rate in men is only 0 to 4 percent.¹¹ Whether these low and unreliable conversion rates are sufficient to meet the needs of most adults is unclear, but we do know they are too low to fulfill the omega-3 requirements of pregnant women and infants, as we'll see in [chapter 15](#). It would make more sense to recategorize EPA and DHA as essential omega-3 fatty acids,¹² because ALA is only required if we don't consume enough EPA and DHA—both of which you can obtain from animal foods (or vegan-friendly algae-derived supplements, if you prefer).



ALL OMEGA-3S ARE NOT CREATED EQUAL

The only type of omega-3 found in plant foods is ALA, which our bodies struggle to convert into EPA and DHA (the omega-3s we actually need), whereas animal foods naturally contain EPA and DHA.

Seed Food Carbohydrates. Seed food carbohydrates generally fall into two categories: simple starches that our gut enzymes digest easily down into glucose, and a variety of complex sugars, starches, and fibers that our gut enzymes can't digest at all.

All plant foods contain indigestible carbohydrates, but within the seed foods, the most abundant example is *raffinose*. Raffinose consists of long strands of simple sugar molecules (glucose, fructose, and galactose) woven together in a pattern impossible for human digestive enzymes to unravel, so it sails through the upper intestine completely intact.¹³ This is good news for colon bacteria, who welcome raffinose into the lower intestine with an enzyme called *alpha-galactosidase* that deftly dismantles it into individual sugar molecules. Our colon cells aren't capable of absorbing sugars, so bacteria proceed to ferment them, breaking them down into short-chain

fatty acids like *butyrate* (that colon cells can burn for energy), and gases like hydrogen, carbon dioxide, and methane. Depending on how much you eat and how sensitive you are, you could experience anything from mild discomfort to severe bloating, cramping, indigestion, nausea, and/or diarrhea.^{[14](#)}

Legumes such as beans, chickpeas, and lentils are relatively rich in raffinose and therefore notorious for causing digestive distress. In fact, the raffinose family of indigestible sugars is the single greatest cause of flatulence in the human diet.^{[15](#)} Soaking, sprouting, fermenting, and cooking can reduce the amount of raffinose in seed foods. Beano®, an over-the-counter anti-gas remedy, contains the same enzyme that your colon bacteria make, so if you swallow this enzyme before you eat beans, it will break the starches down into sugars in your upper intestine where they can be absorbed.

NUTRITIONAL QUALITY: MICRONUTRIENTS

Many seed foods contain B vitamins (especially B1, B2, B6, and B9) and nuts can also provide vitamin E and vitamin K1, but this food group is otherwise relatively micronutrient-poor. Although standard nutrient tables list many seed foods as being high in essential minerals such as magnesium, zinc, iron, copper, or manganese, remember: Just because a food contains a nutrient doesn't mean we can access it.

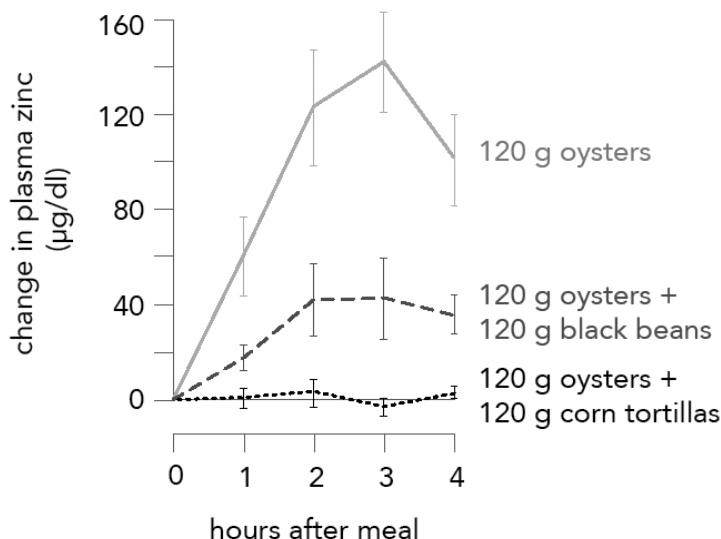
Many Seed Food Minerals Are Hard to Access

All seed foods contain *phytate*, a mineral magnet that binds tightly to iron, zinc, calcium, magnesium, and copper. When the time comes to sprout, phytate gets broken down, releasing these essential minerals to support the growth of young seedlings.

Humans don't possess the enzymes needed to disable phytate and free minerals from its grip, so most phytate survives digestion and makes it all the way down to the colon. Although your colon bacteria will start to break it down, most phytate leaves your system intact, carrying precious minerals away with it.^{[16](#)}

Seed foods can interfere substantially with your ability to absorb

minerals from other foods as well. The graph shown below¹⁷ illustrates the degree to which seed foods blocked absorption of zinc from Atlantic oysters, the richest known source of zinc among all animal foods. In a 1979 experiment, researchers demonstrated that when people ate oysters, their zinc levels peaked nicely in the bloodstream, indicating excellent absorption. When oysters were eaten with black beans, people absorbed only about half the zinc from the oysters, and when oysters were eaten with corn tortillas, people absorbed virtually none of the zinc from the oysters.



EFFECT OF PHYTATE ON ZINC ABSORPTION

Phytate, an antinutrient found in beans, corn, and other seed foods, strongly interferes with zinc absorption from oysters.

N. W. Solomons, R. A. Jacob, O. Pineda, and F. Viteri, "Studies on the Bioavailability of Zinc in Man. II. Absorption of Zinc from Organic and Inorganic Sources," The Journal of Laboratory and Clinical Medicine 94, no. 2 (1979): 335–43.

Phytate can only block absorption of non-heme iron (the only form of iron found in plant foods); it is powerless over heme iron.¹⁸ Remember, animal foods contain a mixture of heme iron and non-heme iron. Therefore, if you consume iron-rich animal foods such as steak, duck, or mussels together with seed foods, you will still be able to absorb the heme iron from those animal foods.

Soaking, cooking, sprouting, and fermentation can all help reduce the phytate content of whole grains.¹⁹ Most grains (except corn) store phytate

in their bran coating, whereas most beans, nuts, and seeds store phytate in their starchy centers. This means that whole grains (which have not had their bran layer removed), such as wheat berries and brown rice, are high in phytate, but refined grains (which have had their bran layer removed), such as wheat flour and polished rice, are low in phytate. This is one reason why whole grains are not always more nutritious than refined grains.

Soy and Millet Interfere with Thyroid Function

The thyroid is a butterfly-shaped gland located in front of your throat. Its main job is to combine the essential mineral iodine with the (nonessential) amino acid tyrosine to make thyroid hormone. Thyroid hormone regulates the metabolism of every cell in the brain and body, directs multiple aspects of brain development, and influences dozens of genes that regulate mood, energy, and sleep,²⁰ which explains why people with low thyroid hormone activity (*hypothyroidism*) can feel sluggish and depressed. When the thyroid gland malfunctions, it can grow large and swollen, forming a *goiter*, therefore any substance with the potential to stress the thyroid gland (whether it ultimately leads to a goiter or not) is called a *goitrogen*. The most important goitrogens in the seed food family are soy *isoflavones* and millet *flavonoids*.

Soy Isoflavones. The isoflavones in soybeans interfere with *thyroid peroxidase*, the enzyme your thyroid gland uses to insert iodine into thyroid hormone. The more isoflavones you consume, the more iodine you will need to overcome their anti-thyroid effects.

For example, it's been known since the 1960s that soy-based formula increases the risk that healthy infants will develop hypothyroidism. For this reason, manufacturers are required to add iodine to soy formula to help counteract the anti-thyroid effects of soy isoflavones,²¹ and pediatricians recommend that children with hypothyroidism avoid soy products.

There is good evidence that soy has anti-thyroid effects in adults as well.²² In an eight-week-long randomized controlled trial, sixty patients with borderline hypothyroidism were given either 2 mg of soy isoflavones per day (the estimated amount in a typical omnivore diet) or 16 mg of soy isoflavones per day (the estimated amount in a typical vegetarian diet). The vegetarian dose of soy isoflavones was three times more likely to cause

patients to convert from borderline (subclinical) hypothyroidism to full-blown (clinical) hypothyroidism.

Since 2009, the Senate Commission on Food Safety of the German Research Foundation has recommended that adults at higher risk, such as people with iodine deficiency, subclinical hypothyroidism, and/or those born with thyroid dysfunction, avoid isoflavone supplements, and limit total isoflavone intake from food to no more than 50 mg per day²³ (the amount found in four to eight ounces of tofu).

Millet flavonoids. The anti-thyroid flavonoids in millet are particularly potent. Depending on where you live, you may know millet as birdseed, a gluten-free alternative to wheat, or an everyday grain. In Sudan and several other countries in Africa, pearl millet is a staple grain that appears to contribute significantly to the high incidence of goiter in children, even in areas where iodine deficiency is not a concern.²⁴ Unfortunately, soaking, cooking, sprouting, and fermenting can't destroy soy or millet goitrogens, so the only way to reduce your exposure is to limit the quantity of these foods in your diet.²⁵

Other Seed Food Antinutrients

Phytates and goitrogens aren't the only antinutrients in seed foods. Cocoa beans and almonds are examples of seed foods high in *oxalates*, which interfere with mineral absorption. Cocoa beans and walnuts are examples of seed foods high in *tannins* which interfere with protein, vitamin B1, and iron absorption. We'll return to these in the next chapter, but now it's time to shift our attention away from antinutrients—hopefully before you notice that cocoa beans (the main ingredient in dark chocolate) are seed foods...

SEED FOOD WARFARE

All plants defend themselves using chemical weapons, and the most heavily defended part of the plant is its seeds, so seed foods contain a multitude of malicious molecules, each designed to harm you in its own unique way.

Lectins and Human Health

When a seed is stressed or damaged (which can occur in the process of our

chewing and trying to digest it), it releases defensive molecules called *lectins* to identify and attack potential enemies. These sticky proteins recognize friend from foe by reading signature carbohydrates on the surfaces of foreign cells. Every living thing is armed with a variety of its own species-specific lectins, some much more potent than others. All plant foods contain lectins, but the highest concentrations of some of the most aggressive plant lectins are found in grains and legumes, most notably kidney beans, fava beans, peanuts (which are technically legumes), peas, soybeans, and wheat.

Why would the lectins in grains and legumes be more vicious than those in nuts and seeds? It may be because nut trees and certain seed-bearing plants need animals to spread their seeds, whereas grain and legume plants don't. If nut and seed-producing plants make their lectins too toxic, they risk harming the animals that help them scatter their seeds. Grains and legumes don't need to concern themselves with the safety of other species; wind currents carry grain seeds to new destinations, and legume pods explode in the heat of the sun, shooting their seeds into the air, so they can make their lectins as toxic as they like. While most edible nuts and seeds can be eaten raw without immediate consequence, lectins are the reason why grains and legumes should never be consumed raw. Documented outbreaks of food poisoning in humans resulting in severe gastrointestinal illness have been caused by undercooked kidney beans and broad beans, as well as raw, unripe French beans and runner beans.²⁶

Lectins resist digestion and can bind to, penetrate, and change the behavior of their target cells in any number of ways. Some lectins cause blood cells to clump together, some stimulate cells to multiply, some poison *ribosomes* (cell protein factories), and some command cells to commit suicide (apoptosis).²⁷ Lectins have been shown in laboratory studies to damage human intestinal cells and in animal studies to poke holes in their intestinal linings, leading researchers to speculate about whether they could contribute to increased intestinal permeability (aka “leaky gut”) in living humans as well.²⁸ Leaky gut syndromes are more common in people with certain psychiatric conditions, including autism, mood disorders, and schizophrenia, although it's unclear how exactly they are related to each other.²⁹ (Note that high blood glucose levels also loosen the connections between intestinal cells, as can anything that causes inflammation).³⁰

Lectins can penetrate the cells of your digestive tract and enter your bloodstream,³¹ which may trigger your immune system to produce antibodies against them. Researchers at UCLA found that approximately 15 percent of healthy blood donors tested positive for antibodies to lectins from wheat, kidney beans, peanuts, peas, soybeans, and lentils. These antibodies bind to a wide variety of human tissues and proteins throughout the brain and body, with wheat lectin antibodies being the most reactive and most promiscuous. The fact that lectins can trigger your body to produce antibodies that cross-react with your own cells lends support to the theory that lectin-rich seed foods may help pave a path to autoimmune diseases—conditions in which your immune system targets your own healthy cells for destruction.³²

Lectins are so skilled at breaching cell boundaries that researchers use them to chaperone drugs and other substances across the gut lining and even across the blood-brain barrier. Rat studies conducted by scientists at Penn State used a lectin isolated from peas to help a pesticide associated with Parkinson's disease pierce the stomach and travel into the brain via the *vagus nerve* (which connects the gut with the brain). Only when the pesticide was accompanied by pea lectin was it able to cause Parkinson's symptoms in these animals, leading the researchers to theorize that "lectins may represent a key environmental factor in the development of this disease."³³

Despite decades of lectin research, human clinical studies are few and far between, so we can only speculate about their potential to contribute to mental health problems. However, it's safe to say that plants do not produce these molecules with your well-being in mind. If the lectins in grains and legumes are poking holes in your gut, provoking your immune system to paint targets on your healthy cells, and even penetrating your brain, they could be harming you in ways that we don't yet fully understand. I encourage you to err on the side of caution and either avoid seed food lectins entirely or at least take steps to minimize their risks.

Fortunately, most lectins can be nearly completely inactivated by pre-soaking seed foods for twelve hours and then boiling for at least fifteen minutes, with some authorities recommending boiling for a full hour. Dry heat (baking or roasting) is not as effective as prolonged boiling, so baked goods made with grain or bean flours are not as safe as boiled goods. For

example, dry roasting only removes about 75 percent of the lectins from raw peanuts. Sprouting and fermentation also reduce lectin content to some extent.^{[34](#)}

Flaxseeds Produce Cyanide

Flaxseeds have become a popular “superfood” in recent years, particularly among those following vegan diets, because they are one of the few plant foods rich in the omega-3 fatty acid ALA. But are they worth the risk?

Sink your teeth into a handful of raw flaxseeds or whirr them into a smoothie and the damage you inflict on them will inspire a harmless chemical inside those seeds called *linamarin* to transform itself into *cyanide*. Cyanide is a deadly chemical that muscles its way into your mitochondria and inserts itself into your electron transport chains, blocking their ability to produce energy.

Fortunately, the liver can detoxify a certain amount of cyanide (a 150-pound individual can detoxify 4.2 mg of cyanide per hour), and the quantity of cyanide in flaxseeds is fairly low (approximately 5 mg per tablespoon). However, these estimates can vary depending on the flaxseed source and preparation method, as well as the health status of the consumer, so there is enough uncertainty surrounding the potential risk that national guidelines vary. For example, the European Food Safety Authority (EFSA)^{[35](#)} warns that toddlers should not consume more than one-third of a teaspoon of flaxseed per day, and that adults should not consume more than about three-and-a-half teaspoons per day, or their blood levels could edge into the toxic (but still non-lethal) range, whereas Food Standards Australia New Zealand (FSANZ) view flaxseeds as generally safe.^{[36](#)}

There are no documented human cases of death (or even illness) related to flaxseed consumption, which is reassuring, but this doesn’t help us understand how repeated exposure to small amounts of cyanide over time may affect the health of your brain mitochondria, and therefore your mental health. If there is any risk involved, it is likely small, but the good news is that flaxseeds contain the wrong kind of omega-3 fatty acids, so there is no need to eat them in the first place.

Gluten, Wheat, and Mental Health

Strictly speaking, gluten is neither a toxin nor an antinutrient—it is simply a protein found in wheat, barley, rye, and triticale (a cross between wheat and barley). Problems with gluten stem from the fact that we lack the enzymes needed to completely break it down into individual amino acids³⁷—an evolutionary clue that we are not properly adapted to nourishing ourselves with wheat and related grains. Just as fragments of partially digested dairy casein proteins can be problematic, so, too, can partially digested gluten fragments.

Gluten poses clear risks to mental health, at least in susceptible individuals. Gluten can cause celiac disease, an autoimmune disease in which the body generates antibodies against proteins in the small intestine. It is well established that psychiatric conditions are more common in people with celiac disease, particularly autism spectrum disorders, attention deficit disorders, depression, anxiety, and eating disorders.³⁸ (About half of people with celiac disease have no gastrointestinal symptoms whatsoever, which often delays the diagnosis for many years.³⁹)

However, even people *without* celiac disease can have heightened immune reactions to gluten. For example, people with schizophrenia, autism spectrum disorder, and bipolar disorder are more likely to have antibodies against gluten-derived peptides (incompletely digested protein fragments) in their bloodstream than those in the general population.⁴⁰ Levels of these antibodies can be up to four times higher in people with schizophrenia compared to people without schizophrenia.⁴¹

There are published case reports of people with schizophrenia and autistic spectrum disorders improving on gluten-free diets,⁴² and several studies suggesting that wheat can contribute to depression and anxiety symptoms in some people.⁴³

The best documented example I'm aware of is a 2015 case report that meticulously details the case of a fourteen-year-old Sicilian girl with severe psychotic symptoms, including hallucinations, paranoia, and suicidal thoughts, whose symptoms were due entirely to non-celiac wheat sensitivity.⁴⁴ A dramatic example, to be sure, but it illustrates how psychiatrically dangerous wheat can be for some people.

I have also personally witnessed a handful of cases of clinical depression which resolved entirely by removing gluten-containing foods from the diet, including this case of severe bipolar depression.

A graduate student in his late-twenties came to me for help with severe bipolar II disorder. When depression overtook him, he would spend most of the day in bed and couldn't read, study, or attend classes. When hypomania arrived, it would infuse the depression with the energy to act on his depressed thoughts, which included thoughts of harming himself. Like some people with depression, he felt compelled to make shallow cuts on the skin of his forearms with a razor, which would temporarily bring some relief. For years, these unpredictable shifts in mood had interrupted his life; he had been hospitalized multiple times with suicidal thoughts and cutting behaviors and was a year behind in working toward his degree due to medical leaves of absence. Having tried more than a half a dozen psychiatric medications that did nothing more than cause unpleasant side effects, he consulted with me about dietary interventions. He didn't feel ready to try a major dietary change such as a paleo or ketogenic diet, so he asked if there was anything simpler he could try first. I recommended he start with a gluten-free experiment, which he eagerly committed to, and within a week both he and his family noticed a major change. His mood lifted, his energy, concentration, and productivity improved, and he felt better than he had in many years. He responded so well to this plan that his whole family adopted the diet in solidarity.

It must be noted that when people feel better on gluten-free diets it can be very difficult to confirm without special testing that the culprit was gluten itself and not some other problematic ingredient in wheat products such as wheat germ agglutinin (a potent lectin) or *glyphosate* (a neurotoxic pesticide⁴⁵ commonly sprayed on wheat crops).⁴⁶ The clinical research in this field is far from conclusive, and most people who simply eliminate gluten-containing foods from their diets won't see their psychiatric symptoms disappear, but if you are suffering from mental health problems, you have nothing to lose by exploring this approach to see if it helps you feel better.

FEAR OF MISSING OUT?

The 2020 U.S. Dietary Guidelines list grains as an essential food group, recommending that adults eat six ounces of grain-based food per day—the equivalent of six slices of bread or three cups of rice.⁴⁷ Why are grains—the

least nutritious of the seed foods—considered essential? Do they contain nutrients we can't obtain anywhere else?

Nutrition policymakers explain that grains belong in a healthy diet because:

- grains are rich in complex carbohydrates and fiber (true, but you can obtain these from fruits and vegetables as well)
- grains contain B vitamins (true, but only a few)
- grains contain essential minerals (true, but most are hard to access)
- grains contain antioxidants and phytonutrients (true, but see [chapter 14](#))

Instead of giving grains their own group, the United Kingdom's Eatwell Guide places grains in a “starchy foods” group, so grains are not explicitly required, although four of the five starchy foods in that group are grains or grain products.⁴⁸ By lumping grains in with starchy vegetables like potatoes, the Eatwell Guide is quietly acknowledging that grains have no nutritional advantages over other starchy foods.

But don't you need to eat whole grains to be healthy? You've heard this countless times, but there is no evidence to support it. In 2019, researchers in New Zealand published a sweeping meta-analysis of fifty-eight human clinical trials, concluding that whole grains can help in the fight against numerous chronic diseases including obesity, cardiovascular disease, type 2 diabetes, and certain cancers. This paper received a great deal of positive attention from scientists and journalists around the world, but examine the authors' conclusion and you may notice the problem: “Implementation of recommendations to increase dietary fibre intake and to replace refined grains with whole grains is expected to benefit human health.”⁴⁹

Do you see what I see?

Researchers didn't compare diets with whole grains to diets without grains; they made the common mistake of comparing diets containing whole grains to diets containing *refined grains*. These studies can't tell us that whole grains are healthy; all they can tell us is that *whole grains are healthier than refined grains*. To my knowledge, there are no studies designed to explore the question of whether we need grains of any kind in

the first place, although it's difficult to imagine why we would, since there are no nutrients in grains that we can't obtain elsewhere.

What about the other seed foods? The U.S. Dietary Guidelines recently placed beans, nuts, and seeds together in the "protein foods" group along with lean meat, seafood, poultry, and eggs,⁵⁰ indicating that you can meet your protein needs by eating seed foods or animal foods, your choice. Grouping these foods together implies that they are nutritional equals, when the fact is that seed foods are nutritionally inferior to animal foods in every way.

SEED FOODS, IN A NUTSHELL

- Seed foods are the most nutritious and the most problematic of all plant foods.
- Seed foods can be good sources of several B vitamins. Fattier seed foods like nuts may also contain vitamin E and K1. Seed foods do contain minerals but most are hard to access due to antinutrients.
- The proteins in most seed foods (with the exception of soy, quinoa, and buckwheat) are incomplete, meaning they do not provide all nine essential amino acids in sufficient quantities, so meeting amino acid requirements without animal foods requires education and planning.
- Grains, beans, nuts, and seeds fiercely protect their embryos using defensive toxins that can jeopardize your gut health, immune system, thyroid health, mitochondrial health, and mental health.
- The defensive lectins in grains and legumes tend to be more toxic than those in most nuts and edible seeds.
- Flaxseeds, chia seeds, and walnuts are good sources of the omega-3 fatty acid ALA, but the omega-3s we actually need are EPA and DHA.
- Flaxseeds contain small amounts of cyanide. It is unclear whether they pose any risk to human health.
- Many seed foods, especially legumes, are high in raffinose, which can contribute to IBS symptoms.

Tips for Nourishing, Protecting, and Energizing your Brain with

Seed Foods

- I encourage everyone to completely avoid all grains and legumes, as their health risks far outweigh their benefits. Grains and beans are also high in carbohydrate, so they are potentially unsafe for people with insulin resistance.
- Nuts and seeds are problematic as well, so I encourage you to explore how these foods affect you.
- If you don't eat animal foods: you will need to rely on soy and other seed foods for protein; see [chapter 15](#) for your best options.
- If you eat soy regularly, your iodine requirements may be higher. Limit soy if you have hypothyroidism.
- If you include grains and/or legumes in your diet, learn preparation techniques to reduce phytate content and inactivate lectins and other toxins, such as pre-soaking and boiling.
- If you have gastrointestinal issues, limit your intake of legumes and nuts, which are very hard to digest. Rice and spelt are examples of seed foods that are more comfortable to digest.
- If you don't eat animal foods, the best (and only) sources of the omega-3s you need are vegan-friendly supplements sourced from algae.
- If you include some animal foods in your diet, there is no nutritional or health reason to include any seed foods in your diet.

I realize it's hard to imagine life without seed foods—particularly grains, which most of us have been eating multiple times a day since early childhood—but the health benefits for your brain and the rest of your body are well worth it. I recognize that my recommendation that you remove grains and legumes from your diet may come across as radical and risky, flying in the face of decades of global health policy and thousands of years of human history. Grains and legumes are trusted and beloved by cultures in all four corners of the globe. Countless people around the world live with food insecurity and depend on starchy grains and legumes for their survival. Wheat, rice, and maize together comprise 60 percent of the world's caloric

intake⁵¹—in some regions by choice, in others by necessity. However, it is important to make a distinction between dietary strategies that ensure the survival of large populations and dietary strategies that *optimize* the health of your brain and the rest of your body. If you are seeking better mental and physical health, eliminating grains and legumes is the single most important change you can make to your whole-foods diet.

CHAPTER 13

Fruits and Vegetables: Distinguishing Friend from Foe

A great many of the chemicals plants produce are designed, by natural selection, to compel other creatures to leave them alone.... But many other of the substances plants make have exactly the opposite effect, drawing other creatures to them by stirring and gratifying their desires.

—Michael Pollan, *The Botany of Desire*

Fruits and vegetables have much to offer. They come in a wonderful diversity of shapes, colors, and textures and lend a delicious complexity of unique flavors to home cooking. Many of them are good sources of several essential vitamins and minerals. Most vegetables that grow aboveground are low in carbohydrate, so they are metabolically safe for people with insulin resistance and well-suited for ketogenic diets. Yet there are limits to their capabilities, and even a few risks to be aware of, so each fruit and each vegetable must be evaluated on its own merits.

Fruits and vegetables are widely viewed as virtuous superfoods, so we're often told that the more of them we eat, the healthier we will be. Packed with vitamins and minerals, bursting with colorful antioxidants, and full of fiber, we imagine them nourishing our souls, steeling us against chronic disease, and sweeping our colons clean. The U.S. Dietary Guidelines advise eating two cups of fruit and two-and-a-half cups of a wide variety of vegetables per day,¹ implying that both fruits and vegetables are necessary, and that all of them are equally good for you. This recommendation, and indeed *every* official proclamation you've ever heard about these foods from "eat the rainbow" to "get your five-a-day" to "most

of us aren't getting enough fruits and vegetables," is grounded entirely in nutrition epidemiology. This doesn't mean that fruits and vegetables aren't good for you; it just means that if we want to understand how these foods affect our health, we need to start fresh with the facts.

WHAT ARE FRUITS AND VEGETABLES?

The words *fruits* and *vegetables* are uttered in the same breath so often that you could be forgiven for thinking of them as having similar biological and nutritional effects, yet nothing could be further from the truth. Some plants want you to eat their fruits, so they go out of their way to make them safe and appealing to you, but no self-respecting plant wants you to eat the rest of its body, so all vegetables, without exception, are *trying* to hurt you. Whether they succeed and how much harm they might do depends on the type of vegetable, how you prepare it, how much of it you consume, and how strong your defenses are.

If It Contains Seeds, It's a Fruit

Fruits are plant ovaries—the core of the plant's reproductive system. Just as human ovaries produce and store eggs, plant ovaries (fruits) produce and store seeds. Unlike human ovaries, fruity ovaries are also responsible for dispersing those seeds, and have evolved a variety of clever strategies for accomplishing this task.² If a plant requires mammals like us to disperse them, it may wrap its seeds in a sweet, succulent fruit, paint it a pretty color, and dangle it seductively from a tree branch, like an apple tree does. When a bear swallows an apple, core and all, the seeds wend their way through her intestines as she moseys about. By the time the seeds exit her digestive tract, she will likely have wandered far from the tree to deposit the seeds onto the ground, complete with a supply of natural fertilizer as a parting gift.

Most of the fruits we eat are relatively nontoxic and easy to digest, therefore safe to consume in moderation unless you have insulin resistance or fructose intolerance (more on this shortly). We think of fruits as being sweet, but many are not. For example, cranberry seeds are housed in bitter, dry, featherweight fruits because they prefer to float on water currents in

search of greener pastures, and therefore don't need to manipulate us into eating them. Some fruits are even downright poisonous. Just one of many examples is the fruit of the manchineel tree, which grows in Florida, the Caribbean, Mexico, and Central America. Sometimes called "the little apple of death," this small green fruit tastes sweet but contains a caustic toxin that causes blistering ulcers and burns.³

Some fruits may want you to swallow and transport their seeds, but the last thing any fruit wants is for you to chew and digest their seeds to death, and every seed has its own clever way of trying to avoid this terrifying possibility. Strawberry seeds are too tiny to chew. Tomato seeds are too slippery. Peach pits are rock-hard and impenetrable. Watermelon seeds and apple seeds are easy to chew but so bitter that some animals swallow them whole while we prefer to spit them out. Smarter still, the flesh of some edible fruits contains sugar alcohols like sorbitol, mannitol, and xylitol, which act as natural laxatives (this is how prune juice works). Sugar alcohols speed seeds through your innards, increasing the chances that they will escape unscathed.

If It Doesn't Contain Seeds, It's a Vegetable

Any plant part that is not a fruit or a seed is considered a vegetable. Cucumbers and eggplants are technically fruits, because they contain seeds, whereas carrot roots, celery stems, and spinach leaves are vegetables. Most of the vegetables we eat are good sources of certain nutrients and low in toxins, but the majority of the world's "vegetables" are considered inedible because they are too bitter or toxic, which is why we rarely drool at the sight of a pile of yard clippings. For example, botanical surveys conducted in England found that only about one-quarter of the plant species that grow in that country contain edible parts.⁴

PLANTS VS. ANIMALS: NATURE'S FOREVER WAR

Traces of fruit and vegetable matter discovered within ancient cooking vessels⁵ and inside the bodies of Danish mummies⁶ tell us that these foods have been on the human menu for at least 10,000 years, but we have probably been eating fruits and vegetables regularly since the dawn of our

species, even if we don't have physical evidence of it. Our closest primate relative, the chimpanzee, eats lots of fruit and some vegetables, too, which suggests that we have probably been doing the same ever since our ancestors began evolving away from chimpanzees seven to nine million years ago.⁷ Several million years of plant-eating experience may seem like enough time to understand them, outsmart them, and safely exploit them for our own nutritional purposes, but plants have been on Earth for *hundreds of millions of years*, allowing them ample time to perfect their chemical weapons arsenal. To date, more than 50,000 defensive compounds have been identified in the plant kingdom, so our prehistoric ancestors had their work cut out for them.⁸ To protect themselves and future generations against plant poisonings, they had to learn the hard way through smell, taste, and experience which plant parts could safely be eaten, and then pass that hard-earned wisdom down through generations. Our bodies have also evolved clever biological coping mechanisms to help us survive the violent plant molecules we regularly encounter. If they hadn't, we wouldn't be able to safely consume most of the plant foods we eat today.

Our first line of defense is aversion to bitterness—nature's way of communicating toxicity. Every baby who spits out a Brussels sprout is simply exercising excellent evolutionary instincts, whereas modern messaging about the health benefits of bitter vegetables such as kale convinces many adults they need to swallow them even though they don't taste good. Many of the fruits and vegetables we eat today have been painstakingly bred to reduce bitterness and/or increase sugar content to make them safer and more appetizing to consumers.⁹

Our second line of defense is our intestinal lining, which intelligently identifies friend from foe and decides which molecules may cross into the bloodstream and which should keep moving along toward the exit.

Any unsavory substances that manage to breach this barrier and enter the bloodstream will immediately encounter our third line of defense: the liver. The largest organ in your body (except for your skin), your liver filters dangerous substances out of your blood, transforms them into harmless waste products, and ships them out of the body—usually through your kidneys and into the urine.

Still, if you eat too many of the wrong plant foods, you can overwhelm all of these defenses. Worse still, if you have poor gut, liver, kidney, or

immune health, your ability to comfortably tolerate even small amounts of certain plant foods (and in some cases, most or all plant foods) may be compromised.

THE NUTRITIONAL QUALITY OF FRUITS AND VEGETABLES

We think of fruits and vegetables as being excellent sources of essential vitamins and minerals, but the list of micronutrients this food family can offer you in any significant amount is fairly short: vitamin C, vitamin K1, vitamin B6, vitamin B9 (folate), potassium, magnesium, manganese, calcium, and *carotenoids* such as beta-carotene, which we can turn into vitamin A. (Other nutrients such as iron can be found in certain fruits and vegetables, but antinutrients restrict our access to them.)

Most fruits and vegetables are high in carbohydrate, low in protein and very low in fat. (Notice that this macronutrient profile is almost exactly the opposite of a ketogenic diet.) The exceptions to the low-fat rule are avocados, olives, and palm fruits, which are so high in fat that we can extract and bottle their oils. Note that these oils come from the flesh of these fruits rather than their pits, so they are not seed oils. Since the majority of fruits and vegetables are low in protein and fat, the major macronutrient they have to offer us is carbohydrate.

Are Carbohydrates Good or Bad?

One of the most striking and important differences between plant and animal foods is that all animal foods, with the exception of milk, are extremely low in carbohydrate, whereas most plant foods are high in carbohydrate. This is because animals store most of their energy as fat, but plants store most of their energy as carbohydrate, usually as underground lumps of starch. Some plants also sweeten their fruits with simple sugars to entice animals to eat them. Therefore, fruits and root vegetables (like potatoes and beets), tend to be particularly rich in carbohydrate.

Just because we don't need to eat carbohydrates doesn't necessarily mean carbohydrates are "bad." In fact, carbohydrates are so "good" that our bodies make glucose and glycogen from scratch to nourish our cells—

especially our brain cells. As omnivores, we can extract nutrients from both plant and animal foods and we can burn both carbohydrates and fat for energy. The unrefined sugars and starches naturally present in fruits and roots should not pose any significant danger to your health—*so long as you are metabolically healthy.*

Unfortunately, most of us have accumulated a significant degree of metabolic impairment and can no longer safely tolerate much carbohydrate of any kind, even from fruits and vegetables. If you have insulin resistance or diabetes, the sugars and starches in fruits and starchy vegetables can contribute to high insulin and glucose levels, and if this is happening for you, it will damage your physical and mental health over time.

Just as with seed foods, fruits and vegetables contain a variety of sugars and starches, some of which we digest easily down into glucose and some of which we can't digest at all. Those we can't digest ourselves wind up in the colon where they are fermented by gut bacteria.

Fermentable Carbohydrates and IBS

Fermentable carbohydrates are also called *FODMAPS* (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols). We are sometimes advised to seek out foods containing resistant starch and certain other FODMAPS to feed our microbiome, but if you have irritable bowel syndrome (IBS), you may want to limit your FODMAP intake.

IBS is common, affecting 10 percent or more of people in North America, Northern Europe, and Australasia.¹⁰ Symptoms can include constipation, diarrhea, bloating, and/or cramping, and people with IBS are two to three times more likely to have symptoms of anxiety or depression than people without IBS.¹¹ We hear all the time that boosting fiber intake by eating more fruits and vegetables is one of the secrets to better digestion, but eating more fruits and vegetables can sometimes cause more problems than it solves, because so many of the carbohydrates they contain are indigestible. Study after study finds that when people with IBS follow a low-FODMAPS diet with the guidance of a dietitian, the majority of them feel much better.¹²

With the exception of lactose (which comes from milk), all FODMAPS come from plant foods, and the ones most commonly found in fruits and

vegetables are raffinose, resistant starch, fructose, fructans, and sugar alcohols.

We learned about raffinose in the last chapter when we discussed legumes, but **asparagus** and **cruciferous vegetables** (such as broccoli, cabbage, and kale) are also rich in raffinose.

As its name suggests, *resistant starches* are starches that resist being digested by our intestinal enzymes. The few fruits and vegetables that contain resistant starch include **plantains, underripe bananas, Jerusalem artichokes, tigernuts, and cooked, cooled potatoes.**

Fructose is nature's sweetener, so the sweeter a plant food is, the more fructose it contains. We absorb glucose extremely well, but fructose can be harder to absorb, especially if it's not accompanied by an equal amount of glucose. Sweet vegetables tend to have a good balance between glucose and fructose, but some fruits contain a lopsided amount of fructose, including **pears, apples, watermelon, cantaloupe, grapes, guava, mango, and papaya.** Any fructose in these fruits that isn't absorbed in the upper intestines will continue down to the colon to be fermented.

Garlic, onions, shallots, artichokes, and cruciferous vegetables contain *fructans* (chains of fructose molecules with a glucose molecule tacked on to one end). We can digest and absorb only about 10 percent of the fructans we consume, so most of them end up fermenting in the colon.¹³

Stone fruits like cherries, plums, peaches, apricots, and nectarines contain small amounts of *sugar alcohols*, which act as natural laxatives. If you overeat these fruits or eat them dried (which concentrates the sugar alcohols), you may experience diarrhea and related symptoms.

Pulp Fiction: The Fiber Fallacy

It is widely believed that the fiber content of fruits and vegetables is key to their health benefits. So what is fiber, and how does it protect us from disease?

Just as bones and cartilage support the bodies of animals, fiber provides architectural scaffolding for plants. There are two types of fiber: *soluble* and *insoluble*, and all plant foods contain a combination of both in varying amounts.¹⁴ We are told that soluble fiber is good for us because it slows things down and that insoluble fiber is good for us because it speeds things

up. Not only are these mixed messages confusing, but they suggest that our gastrointestinal system can't be trusted to regulate its own behavior.

Insoluble Fiber: The Tough Stuff. My grandmother used to affectionately refer to insoluble fiber as "roughage." Insoluble fiber can't absorb water; it's the type of fiber that makes asparagus and celery stalks so tough and stringy. Insoluble fiber passes through our digestive system virtually unchanged, because even our colon bacteria struggle to ferment it. We're told insoluble fiber is good for us because it adds weighty "bulk" to the contents of our intestines, helping to push things along, but if you suffer from constipation, adding more indigestible material to an already clogged system can actually make the situation worse.¹⁵ We're told that insoluble fiber sweeps our innards clean of potential toxins—but there is no evidence that the colon needs any help keeping itself clean,¹⁶ and you don't need fiber to keep things moving, because the muscles that encircle your intestines are constantly in motion, rhythmically propelling everything you swallow toward the exit. You also don't need fiber to scrub your intestinal walls, because the interior lining of your intestines completely replaces itself twice a week.¹⁷ In fact, that scrubbing action could do more harm than good, as insoluble fiber can be so abrasive that the cells lining the colon are forced to produce a layer of mucus to shield themselves from damage.¹⁸

Soluble Fiber: The Swell Gel. Soluble fiber absorbs liquid, causing it to partially dissolve and form a gel. (You can see this happen before your very eyes by stirring a soluble fiber supplement such as Metamucil® into a glass of water.) The ability of soluble fiber to hold water is what allows fruits like apples to be juicy while maintaining their firm shape. There are three main reasons we are advised to consume soluble fiber:

1. **Soluble fiber lowers cholesterol.**¹⁹ Some of the cholesterol you eat becomes physically trapped within soluble fiber. Like bits of fruit cocktail suspended deep within a gelatin mold, these cholesterol molecules can't make contact with your intestinal lining, so they can't be absorbed, reducing the amount of cholesterol that enters your bloodstream. However, in [chapter 4](#) we learned that cholesterol is not a toxin, it's a nutrient. Your body wisely decides how much cholesterol you should absorb; it doesn't need any assistance (or

interference) from fiber.

2. **Soluble fiber can soften blood sugar spikes.** Yes, this swollen gel slows digestion down, which can reduce blood sugar highs by 10 to 20 percent,²⁰ but if your blood glucose is spiking to unhealthy levels, you don't need to eat more fiber, you need to eat less sugar. Instead of increasing fiber to dampen sugar spikes, you can prevent sugar spikes altogether by removing the types of carbohydrate that cause them in the first place.
3. **Soluble fiber helps you lose weight.** This type of fiber swells in the gut, slowing digestion and helping you feel fuller, so you may eat less. It also traps some food particles and prevents them from being digested, reducing the number of calories you absorb.²¹ Clinical trials of soluble fiber and weight loss have been mixed.

Does the Microbiome Need Fiber?

When you eat, you're not just eating for one; you're also eating for the nearly forty trillion bacteria that live in your gut.²² Even though we can't digest and absorb fiber for our own nourishment, we are often advised to eat it for the sake of our microbiome.

Different types of bacteria prefer different foods, so if you eat something a particular species of bacteria likes to eat, that species will flourish and multiply. Certain colon bacteria happen to enjoy fermenting soluble fiber, and one of the molecules they produce in the process is a short-chain fatty acid called *butyrate*. Butyrate nourishes colon cells and protects them against inflammation, leading many microbiome scientists to believe that soluble fiber is essential to good gut health. Does this mean that if you remove soluble fiber from your diet, your colon cells will suffer?

Probably not. First of all, soluble fiber isn't the only thing that stimulates gut bacteria to make butyrate—some bacteria can turn amino acids into butyrate.²³ Secondly, butyrate isn't the only molecule capable of nourishing and protecting your colon cells. Butyrate molecules are almost identical to beta-hydroxybutyrate molecules (ketones). Ketones are an excellent fuel source for colon cells, and have even more potent anti-inflammatory properties than butyrate,²⁴ so if you are in ketosis, your colon cells will be

well fed and well protected by circulating ketones whether you are eating soluble fiber or not.

Fiber and the Gut-Brain Connection

The idea that there is a mysterious megacity of microfauna living inside of us that helps digest our food, protect our gut lining, regulate our immune system, and communicate with our brains has captivated our imagination, and what scientists have already discovered is fascinating.

We know that your gut microbiome and your brain communicate with each other directly via the vagus nerve—the large nerve that connects your brain with your abdominal organs. They also communicate indirectly, via chemical messenger molecules that travel through your bloodstream. We also know that when you change your diet, the number and type of bacteria living in your intestines will change rapidly—usually within twenty-four hours or less, so it's a very dynamic and responsive system.²⁵ However, as compelling as it is to imagine that we might be able to control our moods by controlling this symbiotic swarm, it's important to keep in mind that the microbiome has trillions of microscopic minds of its own, so it's difficult to steer it in any one particular direction.

In 2022, a team of researchers reviewed the existing research on food, the brain-gut-microbiome system, and a selected group of psychiatric disorders including autism spectrum disorder, depression, and cognitive decline, and concluded: “To date there is insufficient evidence from mechanistic human studies to make conclusions about causality between a specific diet and microbially mediated brain function.”²⁶ Translation: scientists don't yet understand how the foods you eat affect your microbiome, your brain, or your risk for neuropsychiatric disorders. Unfortunately, this exciting field is still so young that it can't yet tell us how to eat to improve the health of our inner inhabitants, let alone how our food choices affect the relationship between microbiome health and brain health. So, in the meantime, trust your gut: good digestion shouldn't hurt. If a particular high-fiber food doesn't agree with you, there is no evidence that avoiding that food will endanger your health.

FRUIT AND VEGETABLE TOXINS OF MOST CONCERN

Because we are taught to view all fruits and vegetables as innocent and even virtuous, if we don't feel well, we tend not to suspect them as possible culprits. In fact, many people even double down on their intake of fruits and vegetables in an attempt to feel better, not realizing that this strategy could, in some cases, backfire. By now, you know that all vegetables (and a few fruits) contain defensive chemicals that could potentially harm you. It's not my intent to scare everyone away from eating vegetables entirely; most people tolerate a variety of vegetables quite well. My goal is to raise your awareness and encourage you to keep a curious and open mind when it comes to these foods, just in case some of them may be secretly sabotaging your good faith efforts to optimize your health. In other words, if you are having mental or physical health issues, rather than giving all fruits and vegetables the benefit of the doubt, include them on your list of suspects. A complete discussion of all plant food toxins is beyond the scope of this book, so we'll focus here on the fruits and vegetables most likely to jeopardize mental health.

Nightshades Contain Neurotoxins

Meet the edible nightshades (genus *Solanaceae*):

- **Solanum family:** tomatoes, tomatillos, eggplant, potatoes (all types except for sweet potatoes and yams), garden huckleberries
- **Capsicum family:** peppers (bell peppers, chili peppers, jalapeños, pimentos, pepperoncini, paprika, cayenne, tabasco, etc.)
- **Lycium family:** goji berries, wolfberries

At first glance, many of these foods look completely unrelated to each other, but look closely and you'll notice the family resemblance: all nightshade fruits wear the same jaunty little green elfish hat. (Potatoes don't sport these hats, but the fruits of potato plants do.) Nightshade foods are not to be confused with "deadly nightshade" and its poisonous relatives which belong to a different branch of the nightshade family tree.

Nightshades produce *glycoalkaloids*—pesticides that defend against bacteria, fungi, viruses, and insects. Glycoalkaloids are membrane

disruptors. Like tiny hand grenades, glycoalkaloids attack the cholesterol within membranes, causing cells to leak or even burst open on contact.

Glycoalkaloids are also neurotoxins that work exactly the same way nerve gas does—by blocking *cholinesterase*, the enzyme responsible for breaking down acetylcholine, a neurotransmitter that carries signals between nerve cells and muscle cells. With this enzyme blocked, acetylcholine accumulates in synapses and overstimulates muscle cells, which (depending on the type and dosage of the glycoalkaloid) could lead to seizures, paralysis, respiratory arrest, and even death. Glycoalkaloids also cross the blood-brain barrier, where they can influence the alertness, attention, and memory circuits that rely on acetylcholine.

Potato glycoalkaloids are highly toxic and can reach dangerous levels in sprouting, green, rotting, damaged, or unripe potatoes. Well-documented outbreaks of potato-related glycoalkaloid poisoning in schoolchildren have led to serious symptoms such as vomiting, fever, diarrhea, and several instances of coma, along with multiple psychiatric symptoms including extreme restlessness, confusion, and even hallucinations.²⁷

Eggplant can also contain significant quantities of glycoalkaloid—and the more bitter the eggplant, the more glycoalkaloid it contains.²⁸ **Green tomatoes** are chock full of glycoalkaloids, but levels drop dramatically as they mature in the sun, so ripe **red tomatoes** contain only very small quantities. Levels in **peppers** are also quite low,²⁹ and **goji berries** contain only trace amounts.³⁰ Even so, a few of my nightshade-sensitive patients react poorly to these fruits.

Glycoalkaloids are hardy and survive most cooking and processing methods, so the only way to reduce your exposure is to avoid them. Since potato glycoalkaloids are located in the skin, peeling potatoes and removing any greenish areas, “eyes” (budding sprouts), and damaged areas removes virtually all of their glycoalkaloid content, whereas eggplant glycoalkaloids are located mainly in the seeds and flesh, so peeling doesn’t help. Glycoalkaloids are absorbed into the circulation and can take many days to clear, so if you consume too many nightshade foods too frequently, glycoalkaloids may accumulate in your system over time.³¹

Peppers of all kinds contain *capsaicinoids* in and around their seeds to protect them from insects, parasites, and molds.³² (Their sharp taste is also supposed to prevent mammals like us from eating them, but most humans

quite enjoy their spiciness!)

The most familiar member of this fiery family is *capsaicin*, the active ingredient in pepper spray and pain relief creams. Outside the brain, capsaicinoids bind to pain receptors on nerve endings, generating a temporary burning sensation followed by a long period of numbness. These peppery molecules can also cross the blood-brain barrier and become concentrated in the brain and spinal cord. Studies in animals generally find that reasonable amounts of capsaicin are nontoxic, but when consumed too frequently or given in large doses, capsaicin can cause seizures and can even kill neurons by destroying their mitochondria.³³

Most clinical research focuses on capsaicin's potential health benefits rather than its potential risks,³⁴ but extreme exposure situations in the real world such as spicy food eating competitions and overdoses in children have led to severe pain, nausea, vomiting, high blood pressure, heart attacks, and several deaths.³⁵ Several of my more sensitive patients report burning sensations (most often in the soles of the feet), heartburn, and irritability after eating nightshades of any kind, although peppers seem to bring on the most severe episodes. Hot peppers are central to many cuisines around the world, and it is possible that most people tolerate them relatively well, but if you have a mental health concern of any kind, I would encourage you to experiment with a nightshade-free diet to be absolutely sure that these foods aren't preventing you from feeling your best.

Nightshades Also Contain Lectins

The only fruits and vegetables that contain significant quantities of lectins are the nightshades, giving you one more reason to consider avoiding them. You can minimize your exposure by boiling nightshades before you eat them, or by peeling them and removing their seeds (because lectins are located primarily in their skins and seeds).

Cassava Produces Cyanide

Cassava root is a hardy underground vegetable that comes in both bitter and sweet varieties. The sweet variety is the main ingredient in tapioca, and flour made from sweet cassava is becoming increasingly popular as a gluten-free alternative to wheat flour, so you may have noticed new

products like cassava chips making an appearance on the shelves of your local health food store. More importantly, sweet cassava is a staple food for hundreds of millions of people in Africa, Latin America, Southeast Asia, and the Caribbean.

Unfortunately, like the flaxseeds we examined in the previous chapter, fresh cassava root contains linamarin, which turns into cyanide when the root is damaged or chewed—and even sweet cassava contains much higher quantities of linamarin than flaxseeds, so *cassava root must never be consumed raw*. To make matters worse, in the process of trying to protect itself, your body converts cyanide into *thiocyanate*, because thiocyanate is far less toxic than cyanide and easier for your body to eliminate. However, thiocyanate interferes with your thyroid gland's ability to make thyroid hormone, so linamarin is both a mitochondrial poison and a goitrogen.

The WHO considers foods containing cyanide concentrations of up to 10 parts per million (ppm) safe to consume.³⁶ However, fresh sweet cassava root can contain up to 100 ppm, and while peeling and boiling can reduce its cyanide content, it does not remove it completely. In fact, most methods aimed at detoxifying cassava, including cooking, sun-drying, grinding, and fermentation, are incomplete and unreliable.³⁷

If you're wondering how much cyanide might be lurking within those trendy new cassava products in your health food store's snack aisle, you'll be pleased to know that scientists at Australia's Monash University went to the trouble of measuring the cyanide content of twenty-one cassava-based products purchased from markets in the Melbourne area.³⁸ Highly processed tapioca-based products such as chips and flours contained negligible levels of cyanide, as did all cassava chips manufactured in Australia. However, all cassava chips imported from other countries contained cyanide quantities well above the 10 ppm limit. On average, imported cassava chips contained 96 ppm of cyanide, with one brand containing 148 ppm. Some countries (including Australia and New Zealand) have officially adopted the 10 ppm limit for cassava products while others have not, so the amount of cyanide in products in your local area will depend partly on your government's regulations.

While most cassava consumed around the world is of the sweet variety, there are still some places where people resort to bitter cassava to help sustain themselves. Bitter cassava contains up to 2,000 ppm of cyanide and

is therefore extremely toxic.³⁹ Outbreaks of cyanide poisoning caused by bitter cassava have occurred from time to time. In 2017, the Centers for Disease Control (CDC) reported an outbreak in Uganda (where cassava is a staple crop for 57 percent of the population) that affected ninety-eight people; thirty-three were hospitalized and two people died.⁴⁰ Heavy reliance on bitter cassava in some areas of sub-Saharan Africa is causing devastating neurodevelopmental, psychiatric, and neurodegenerative diseases with serious, irreversible symptoms including cognitive impairment and paralysis.⁴¹

If you have a choice, I strongly recommend you completely avoid all cassava products, whether bitter or sweet.

Cruciferous Vegetables Can Interfere with Thyroid Function

Cruciferous vegetables such as broccoli, Brussels sprouts, and kale come in so many varieties that they tend to dominate the produce aisle (see [chapter 17](#) for a complete list).

When cut, bruised, or chewed, crucifers produce natural pesticides called *isothiocyanates* they can use to attack and kill bacteria, fungi, insects, and worms.⁴² These same molecules also behave as goitrogens (which interfere with your ability to make thyroid hormone), although their anti-thyroid effects are considerably weaker than those of soy and millet. Of the crucifers that have been tested, those with the strongest anti-thyroid activity include Brussels sprouts, collard greens, and Russian/Siberian kale. Cooking crucifers will reduce your exposure to their goitrogens,⁴³ so most cruciferous vegetables are unlikely to cause thyroid problems unless you consume them raw in large quantities.⁴⁴ Nevertheless, if you have hypothyroidism or iodine deficiency, limiting your exposure may be wise.

Miscellaneous Toxins and Antinutrients

There are many other risky chemicals lurking within fruits and vegetables, including the ones listed below. These miscellaneous substances are less likely to cause significant issues unless you consume them in great quantities or are prone to sensitivities.

Toxin	Health Risk	Food Examples
Oxalates	Interfere with mineral absorption and can crystallize, contributing to kidney stones in susceptible individuals	Starfruit, spinach, rhubarb, beets, raspberries
Tannins	Bind to and irritate the gut lining, reduce thiamine and iron absorption, and interfere with digestion	Tea leaves, grapeskins, cranberries, pomegranates
Coumarins	Block a liver enzyme responsible for processing a wide variety of medications; increase susceptibility to sunburn	Cinnamon, citrus peel, green tea
Salicylates	Neurotoxic (at very high doses)	Apples, grapes, avocados, citrus, many others (most fruits contain salicylates)
Thiosulfinates	Interfere with clotting system	Garlic, onion, leeks, scallions
Saponins	Disrupt membranes and interfere with digestion	Licorice root, alfalfa sprouts, ginseng
Cucurbitacins	Make tiny blood vessels leaky, which can cause vomiting and gastrointestinal bleeding	Squash, zucchini, watermelon

If this topic feels overwhelming or confusing, don't worry. I've boiled fruits and vegetables down into a special list of what I call the "kinder, gentler" plant foods, which you'll see in the Quiet Paleo food list in [chapter 17](#). Every recipe in this book is designed around that list so you can feel what it's like to eat in a quieter way.

KNOW YOUR WAY AROUND THE RAINBOW

- Some fruits and vegetables are good sources of vitamin C, vitamin K1, vitamin B6, vitamin B9 (folate), potassium, magnesium, manganese, calcium, and/or beta-carotene.
- Many people tolerate a wide variety of fruits and vegetables, while

others are more sensitive.

- Some fruits and vegetables are high in FODMAPS, which can contribute to IBS symptoms.
- All fruits and vegetables contain defensive chemicals, but the ones of most concern are:
 - Nightshades, which contain neurotoxins and lectins
 - Cassava, which produces cyanide
 - Cruciferous vegetables, which have anti-thyroid properties

Tips for Nourishing, Protecting, and Energizing your Brain with Fruits and Vegetables

- Fruits and vegetables highest in nutrients include cruciferous vegetables, dark green leafy vegetables, beets, avocado, pumpkin, and butternut squash.
- Vegetables lowest in nutrients include iceberg lettuce, celery, cucumbers, radishes, and white potatoes.
- Most sweet fruits are nontoxic and easy to digest, so you can enjoy them in moderation as part of a healthy diet, unless you have insulin resistance.
- Sweet fruits and starchy vegetables are high in carbohydrate, so if you have insulin resistance, you may need to focus on non-starchy vegetables and low-sugar fruits to keep your blood sugar and insulin levels in a healthy range
- If you have digestive issues, eating more fruits and vegetables than your system can process could cause more gut health problems than it solves. Cooking, limiting portion sizes, and sticking with lower-FODMAP choices can all improve digestibility.
- Avoid cassava and nightshades, and go easy on raw cruciferous vegetables.
- Cooking reduces many toxins, but some nutrients (especially the B vitamins and vitamin C) are destroyed by heat or leach out into boiling water.

Fruits and vegetables can be delicious, nutritious components of a healthy diet, but pay close attention to how these foods affect you. We know far too little about any negative effects they may have on mental and physical health, because most clinical studies in humans downplay, ignore, or deny their risks, choosing to focus on the potential benefits of this food group. Strong beliefs about the health benefits of “eating the rainbow” can influence not only the hypotheses that researchers choose to test in clinical trials, but also how we interpret their findings. This phenomenon is one of the forces responsible for the rise of the superfood.

CHAPTER 14

Superfoods, Supplements, and the Antioxidant Myth

Where there is life there is wishful thinking.

—Gerald Lieberman

To nourish your brain, you need to choose foods capable of providing you with essential nutrients. To energize your brain, you need to keep your glucose and insulin levels in a healthy range. But what about *protecting* your brain? Is it enough to eliminate damaging ultraprocessed ingredients like refined carbohydrates and avoid plant foods like wheat and cassava that harbor the most egregious toxins? Or must you also include specific foods to bolster your immune system against cancer, dementia, and other serious chronic illnesses? In other words, do some foods offer unique disease-fighting benefits that go beyond their nutrient content?

Countless nutrition epidemiology studies have reported that the healthiest dietary patterns in the world are the ones that contain the greatest quantity and diversity of plant foods.¹ Rather than questioning or testing the validity of these claims, most experimental scientists simply accept them at face value and skip ahead to ask *how* plant foods work their (alleged) magic in the human body. The unsupported belief that plant foods chivalrously shield people from harm has led serious academics, the food industry, and supplement companies on a decades-long quest to identify the ingredients within fruits and vegetables that make them uniquely beneficial to human health.

At first, researchers focused their attention on a few nutrients that are more plentiful in plant foods than in commonly consumed animal foods: vitamin C, vitamin E, and beta-carotene (which we turn into vitamin A).

Unfortunately, when these micronutrients were put to the test in human clinical trials, they fell flat:

- Vitamin C failed to lengthen the lives of people with cancer or prevent a second heart attack.²
- Vitamin E failed to prevent heart disease, and at higher doses *shortened* life.³
- Beta-carotene *increased* the incidence of lung cancer in male smokers.⁴

The lesson to be learned was that vitamins are necessary, but taking more than we need can throw our systems out of balance and lead to unintended consequences.

The vitamin trail having gone cold, researchers found themselves in need of a new plant superhero. In the 1990s, when new technology emerged that allowed scientists to quantify the antioxidant activity of foods and food ingredients, the belief was born that the secret to the health benefits of fruits and vegetables was that they contained special *phytochemicals* with powerful antioxidant properties.

Since the term “phytochemical” simply means “plant chemical,” thousands upon thousands of plant molecules fall into this category. (The term “phytonutrient” is often used interchangeably with the term “phytochemical,” despite the fact that only a few of them are actually essential nutrients.) Phytochemicals have become the most popular *theoretical* answer to the question of why we are supposed to eat a wide variety of plant foods.

The phytochemicals that scientists have studied most closely are *polyphenols*, which are found in all types of plants and come in thousands of shapes and sizes, each with its own unique purpose. Plants use polyphenols to protect themselves from the sun’s radiation, defend themselves from predators, and paint their fruits beautiful colors. You may be familiar with some of the terms used to describe polyphenol families such as flavonoids, isoflavones, tannins, and anthocyanins, or you may have heard of specific polyphenol molecules such as resveratrol or curcumin.

In 2004, the USDA blew wind into the sails of the antioxidant theory by

creating an online database that ranked specific foods according to their *oxygen radical absorbance capacity* (ORAC)—a laboratory measure of their antioxidant activity. Superfood and supplement marketers could then proudly point to the ORAC values of their products as evidence of their special health-promoting properties, and nutrition authorities could weave the antioxidant narrative into public policy campaigns such as “eat the rainbow.”⁵

However, in 2012, the USDA removed the database from its website due to “mounting evidence that the values indicating antioxidant capacity have no relevance to the effects of specific bioactive compounds, including polyphenols, on human health.” They went on to state:

ORAC values are routinely misused by food and dietary supplement manufacturing companies to promote their products and by consumers to guide their food and dietary supplement choices.... The data for antioxidant capacity of foods generated by in vitro [test-tube] methods cannot be extrapolated to in vivo effects [effects in living beings] and the clinical trials to test benefits of dietary antioxidants have produced mixed results.⁶

Translation: Just because a food molecule behaves as an antioxidant in a test tube does not mean it behaves as an antioxidant in the human body.

Like a tree falling in a remote forest, the USDA’s official take-down of the antioxidant theory made no sound. Scientists and the public have only become more enamored with polyphenols and other non-nutrient phytochemicals, unaware that their potential health benefits are rooted almost entirely in unscientific guesswork.

With the exception of nutrients like vitamin C and folate, the vast majority of phytochemicals do not appear to be essential to our health. How can we tell the difference between an essential nutrient and a garden-variety phytochemical? As a 2009 review article puts it: “Whereas the body has specific mechanisms for the accumulation and retention of vitamins, in contrast, phytochemicals are treated as non-nutrient xenobiotics [foreign substances that shouldn’t be there] and metabolized so as to eliminate them

efficiently.”⁷

Translation: Nutrients are welcomed into our bodies as honored guests and invited to stay, whereas phytochemicals are treated as unwelcome intruders to be forcibly removed from the premises. Essential nutrients are absorbed into the bloodstream and welcomed into our cells, often with the assistance of special receptors designed exclusively for them, whereas we have no such receptors for other phytochemicals. Most phytochemicals suffer from poor bioavailability, meaning they are either difficult to absorb or treated as toxins by the liver, which immediately neutralizes them and ships them to the kidneys for disposal.⁸ Essential nutrients have clearly defined roles in the metabolic reactions required for human life, therefore if we lack any one of them, we become seriously ill or die. In contrast, the biological benefits of non-nutrient phytochemicals are purely hypothetical. Ironically, perplexed scientists striving to understand the biological relevance of phytochemicals use the lack of solid evidence in the field as a reason to consume them.

The article concludes: “As yet, claims for benefit outstrip understanding, and some claims have clearly been based on methodology that has severe limitations. Nevertheless, we remain optimistic that there is a real possibility that some dietary polyphenols will be shown to contribute positively to health and well-being, but at the present state of knowledge feel that the only sound advice that one can give is to have as much variety as possible, so as to maximise the range of phytochemicals that are consumed.”⁹

Translation: There’s no solid evidence that phytochemicals are good for you, yet we continue to believe they might be, so eat as many as you possibly can, just in case.

ARE THERE ANY BRAIN SUPERFOODS?

If there’s even a small chance that eating a rainbow of plants might protect us from dreadful diseases like dementia, why not play it safe? Marketing professionals understand our desire to take every precaution and prey on our health hopes and fears, encouraging us to view the plant foods they sell not merely as foods (sources of essential nutrients), but as superfoods with superpowers that supercharge our health. Let’s take a closer look at the ones

that have skyrocketed to brain food superstardom: blueberries, dark chocolate, and red wine.

Blueberries burst onto the superfood scene in the mid-1990s. Upon receiving notice that wild blueberries ranked number one on the ORAC scale of antioxidant activity, the Wild Blueberry Association of North America (WBANA) seized on the opportunity to give their marketing strategy a makeover. The executive director of the WBANA at the time recalls that their previous strategy had been “trying to sell blueberries because they tasted good inside of muffins... Health wasn’t even on the radar screen.”¹⁰ Picking through the history of this transformational moment, *Outside* journalist Doug Bierend went on to write:

Savvy promotion of the fruit was about to help usher in an era of health food obsession that we’re still living in today. No longer mere tasty treat or part of a balanced diet, blueberries would become known as cancer combatants, inflammation interceptors, defenders of cognitive function—each berry a nutritional Navy SEAL.... A superfood was born.¹¹

Blueberry consumption in the United States rose by 599 percent between 1999 and 2014,¹² and when the blueberry was promoted as a superfood in the UK, sales doubled in just two years’ time.¹³

The blueberry’s superpower supposedly lies within purplish-blue polyphenols called *anthocyanins*. However, these pretty pigments suffer from poor bioavailability: Approximately one-third of them are destroyed in our digestive tract, and most of what we are able to absorb is rapidly eliminated from the body.¹⁴ A 2019 review article of twelve human RCTs concluded that studies examining the effect of blueberry consumption on cognition and mood returned mixed results,¹⁵ and that the research was difficult to interpret. Scientists rarely use whole blueberries such as you would see in a grocery store. Instead, they use freeze-dried blueberries, blueberry extracts, blueberry juice, and blueberry powders mixed into sugar water, sodas, juice, or milk, and these blueberry interventions aren’t always compared to appropriate controls. For example, in one study, blueberry concentrate was compared to a (non-alcoholic) fruit “cordial” containing

sugar and two artificial sweeteners.¹⁶ The authors concluded that while their review provided “limited support for the use of blueberry interventions, this should not be interpreted as conclusive evidence for a lack of efficacy but rather that further trials are required to resolve existing limitations.”¹⁷ In other words, just because we can’t prove it, doesn’t mean it’s not true.

So, are blueberries any healthier than other fruits? The main micronutrients in blueberries are vitamin C, vitamin K1, and manganese, but it’s easy to find fruits that outperform blueberries with respect to all of these. Strawberries, for example, contain seven times more vitamin C and half the sugar.¹⁸ Blueberries are lovely and delicious, but there is no evidence that they offer you any unique health benefits.

Dark chocolate’s rise to superfood status also began in the 1990s when Norman Hollenburg, a professor of radiology at Harvard Medical School, observed that the Kuna Indians who lived off the coast of Panama had significantly lower rates of heart disease, type 2 diabetes, stroke, and cancer than their neighbors on the mainland. Noting that these islanders consumed “a 10-fold higher amount of cocoa-containing beverages, 4 times the amount of fish, and twice the amount of fruit” than their less healthy counterparts across the water, his research group zeroed in on cocoa as a “candidate for further study.”¹⁹ Finding that Kuna cocoa was far richer in certain bitter-tasting *flavanols* (polyphenols) than commercially available cocoa products (which, incidentally, had been processed to *reduce* flavanol content to make them more palatable to consumers.)²⁰ Hollenburg teamed up with Mars, Inc. to develop a special flavanol-rich cocoa product to test in clinical trials. Their first study, conducted in 2003, found that flavanol-rich cocoa improved brain blood flow in healthy young adults as well as in healthy individuals in their seventies and eighties.²¹

Since then, Mars and other chocolate manufacturers have funded numerous studies exploring the potential benefits of cocoa on thinking and memory in aging adults. It’s important to understand that most studies do not use dark chocolate itself, and the few that did found no brain health benefits. Most researchers use proprietary flavanol-enriched cocoa powders mixed into a drink or capsules containing flavanol-enriched cocoa extracts. Some of these high-flavanol cocoa experiments have seen improvement in cognitive tests and others have not.²²

We absorb about 80 percent of the cocoa flavanols we consume, but we then immediately begin transforming them into other molecules, and it's unclear whether they can cross into the brain.²³ However, they may not need to cross into the brain, because some of their benefits may be related to their ability to dilate blood vessels, which improves blood flow to the brain. The better your brain circulation, the more oxygen and nutrients your brain receives.

There is no evidence yet that eating dark chocolate itself improves cognition or memory, but what if you are an optimist at heart, or just looking for a reason to eat more chocolate? The dosage of flavanols used in these studies was between 500 and 900 milligrams per day; is there a way for you to obtain that dosage through chocolate alone? When a team of scientists at the University of Reading tested forty-one chocolate bars, they discovered that their flavanol content varied tremendously, from a mere 0.1 milligrams per gram to 3.2 milligrams per gram—more than thirty times more.²⁴ All of the dark chocolate bars were higher in flavanols than all of the milk chocolate bars, but among the dark chocolate bars, there was no relationship between the percentage of cocoa in each bar and its flavanol content. So, there's no way for you to know how much flavanol is in your favorite bar. The only benefit to choosing bars with higher cocoa percentages is that they usually contain a lot less sugar. (Remember that sugar is a powerful promoter of oxidative stress, so if you eat chocolate, be sure to choose low-sugar brands.)

If you are lucky and your favorite brand happens to contain the highest concentration of flavanols (3.2 milligrams per gram), it would take 156 grams of that chocolate—about one and a half bars, depending on the size of the bar—to reach the minimum dose of flavanols used in these studies. However, if you are unlucky and your favorite brand contains the lowest concentration (0.1 milligrams per gram), it would take 5,000 grams to reach the minimum dosage—that's fifty chocolate bars a day. A tall order for even the most ardent chocolate lover.

Red wine is believed to provide cardiovascular and brain benefits due to the presence in red grape skins of *resveratrol*—a polyphenol with antioxidant properties... and a fungicide that grapevines produce to fight gray mold. Should gray mold dare to encroach upon a grape, resveratrol begins methodically dismantling it from the inside out until “no

recognizable cellular structures are visible except ghosts of mitochondria.”²⁵

Resveratrol joined the antioxidant party in 1997 when scientists discovered it could slow the growth of skin tumors in mice. The idea that a cancer-fighting chemical could be found in red wine was intoxicating to the public and sent red wine sales soaring.²⁶ Since then, hopeful researchers have tested resveratrol against all manner of human ailments, including Alzheimer’s disease, albeit with disappointing results.²⁷ Several year-long clinical trials of resveratrol (at 500 to 2,000 milligrams per day) in people with mild to moderate Alzheimer’s disease found no cognitive benefits, and one study even documented significant brain *shrinkage*.²⁸

Notice that all of these studies use resveratrol, when what you are probably most curious about is red wine. Unfortunately, there are no studies testing the effects of red wine on people with cognitive impairment or Alzheimer’s disease. There are many reasons for this, not the least of which is that the quantity of red wine you would need to drink to achieve even the *lowest* dosages of resveratrol used in clinical trials is 500 bottles per day. A typical bottle of pinot noir contains less than one milligram of resveratrol,²⁹ and that lonely milligram of resveratrol is swimming in a sea of alcohol, a powerful promoter of oxidative stress³⁰—the very thing scientists are trying to fight with resveratrol.

There is no question that binge drinking (defined as four or more drinks within two hours for women and five or more for men) and regular heavy drinking (defined as more than one drink per day for women and more than two drinks per day for men) is dangerous for physical and mental health. The WHO estimates that the harmful use of alcohol is a causal factor in more than two hundred medical conditions and that it is responsible for more than three million deaths every year worldwide. But what if you keep your alcohol intake below this limit? Is it safe, or perhaps even healthy to have a glass of red wine with dinner?

Every molecule of alcohol that enters your liver is treated as a top priority toxin.³¹ As soon as your liver sees alcohol coming in, it stops tending to other important business such as making glucose for your bloodstream and burning fat for energy so that it can focus on ridding itself of that alcohol. These metabolic derailments can cause abnormal fat production and storage inside the liver, which can eventually lead to

alcoholic fatty liver disease in people who drink too much. The process of detoxifying alcohol subjects mitochondria to damaging inflammation and oxidative stress—not only in your liver, but in your brain as well.³²

How did alcohol—a toxic, addictive liquid notorious for impairing coordination, judgment, and memory—become associated with dementia prevention in the first place? Essentially, because a group of nutrition epidemiologists enamored with the cuisine and culture of the Northern Mediterranean had observed that the peoples of this region appeared to be healthier than Americans, noticed that red wine was a part of their lifestyle, and assumed (perhaps even hoped?) that the presence of red wine must be partly responsible for their better health.³³ And so it was that when Professor Walter Willett first unveiled their vision of the Mediterranean diet food pyramid in 1993, it featured a grain-heavy triangle with a glass of red wine perched next to it.³⁴ The assumption that red wine contributes to the health benefits of the Mediterranean diet, which has never been tested in human clinical trials, has been misleading scientists, policymakers, and the general public for more than thirty years.

Red wine is not a brain-healthy beverage. If you have a mental health issue of any kind and you currently drink alcohol at any level, I recommend you stop drinking for at least thirty days so that you can evaluate how your current use may be affecting your mood, sleep, concentration, productivity, and overall well-being.

FOOD AS MEDICINE? THE CURIOUS CASE OF A CRUCIFEROUS CRUSADER

How is it that molecules our bodies don't need and often explicitly reject continue to be prized as health superheroes? The confusion is understandable, because ancient wisdom tells us that some plants have medicinal properties, and that is absolutely true.

Plant chemicals can be simultaneously threatening and therapeutic, and *sulforaphane*—an isothiocyanate produced by broccoli plants—is a good example of this apparent paradox.

When broccoli is sitting peacefully in a field, it contains no sulforaphane. This pungent molecule is so toxic to living cells (including broccoli cells) that the plant stores the two ingredients needed to make it in

separate compartments to keep itself safe. However, if broccoli is cut or chewed, the compartments break open and the two ingredients mix together, creating the “mustard bomb” of an insecticide that is sulforaphane.^{[35](#)} Sulforaphane penetrates the insect’s gut lining and begins dismantling the vital proteins it needs to grow and thrive.

Type the word “sulforaphane” into any search engine and you’ll encounter pages of articles hailing its anti-inflammatory and antioxidant abilities which enable it to kill cancer cells, protect the brain from damaging oxidative stress, and treat mental health conditions. Yet sulforaphane itself is not an antioxidant and is powerless against inflammation. After all, it is a chemical weapon designed to *damage* cells, not protect them, and your body knows this. Although we absorb about 75 percent of the sulforaphane we encounter, as soon as it enters our cells, our own internal antioxidant system springs into action binding, neutralizing, and ejecting it from the body as fast as possible, so within about nine hours, it’s all gone.^{[36](#)} It is well known that any benefits shown in cancer prevention studies, for example, are not directly due to sulforaphane itself, but rather to its ability to spur our own healthy defense mechanisms into action.

Sulforaphane can also cross the blood-brain barrier, and emerging research suggests that it can be helpful in certain psychiatric conditions. For example, a six-week RCT in sixty-six patients with a history of heart disease found that sulforaphane tablets reduced symptoms of mild to moderate depression more than placebo.^{[37](#)} A growing number of studies also demonstrate benefits in autism spectrum conditions in children and adults.^{[38](#)}

If sulforaphane extracts can reduce symptoms of autism and depression, does that mean that broccoli, eaten as a whole food, is good for your mental health? This is a common leap in logic, but just because a concentrated plant toxin can help fight existing disease doesn’t necessarily mean that eating that plant food regularly makes you healthier or prevents disease. To use an extreme example, chemotherapy drugs can kill cancer cells but that doesn’t mean that taking a low dose of chemotherapy every day will prevent cancer. There are no clinical studies exploring whether eating broccoli itself on a regular basis can prevent or treat psychiatric conditions.

Sulforaphane isn’t a food, it’s a medicine extracted from broccoli, and all medicines, whether from foods or from pharmaceutical companies, come

with risks, so you should use them only if you really need them. For example, there are studies showing that sulforaphane can overstimulate defensive pathways, tip the scales too far in the wrong direction,³⁹ and *promote* cancer growth.

I suggest that the concept of “food as medicine” may need to be reconsidered. The purpose of food is to *support* normal human biological functions by providing molecules that nourish and energize us. The purpose of medicine, whether it comes from a food or a factory, is to *interfere* with normal human biological functions, and should therefore only be considered if you have an illness that requires a targeted intervention. This blurring of the lines between food and medicine is what feeds our wishful thinking about superfoods and lines the pockets of the superfood industry.

THE ALLURE OF THE SUPERFOOD

What makes a food a “superfood”? In a word, marketing. There is no scientific or legal definition for the term, so anyone can attach the term to any food product, without having to justify it. In fact, ambiguous health claims may enhance the appeal of superfoods, lending them an air of mystery that transcends scientific explanation. In 2022, *GlobeNewswire* reported that the global superfoods market was expected to grow from US\$ 172 million to US\$ 288 million between 2020 and 2027: “In terms of product, Super Fruits is the largest segment, with a share about [sic] 45 percent. And in terms of application, the largest application is Beverage, followed by Bakery and Confectionery Products, Snacks, etc.”⁴⁰

Notice that most superfoods are not whole foods; they are sugary drinks, baked goods, and snack products made with superfood ingredients. Even if a whole food like the pomegranate did contain absorbable polyphenols capable of squelching free radicals in the human body (which they do not), juicing them into curvaceous eight-ounce bottles and then adding 32 grams of sugar—a powerful promoter of oxidative stress—more than drowns out their alleged superpowers.⁴¹

Despite decades of scientific dead ends, we continue to believe that colorful plant foods can rescue us from dementia and other modern diseases—diseases driven largely by poor-quality diets and other unhealthy aspects of our modern lifestyle. On some level, we must know that the secret to

robust brain health doesn't lie in eating more chocolate and drinking more wine, but if it's not true, we're going to have to face some difficult realities and make some hard choices. We want to believe in the power of addition. Adding a puckery fruit like cranberry, a bitter vegetable like kale, or a pill containing extracts of all the fruits and vegetables you don't have time to eat is easy. Addition feels proactive, positive, and empowering, but the truth is that protection is not about addition, it's about subtraction. We already know a great deal about what causes excess inflammation and oxidative stress, and the prime dietary suspects are high refined carbohydrate intake, refined vegetable oils, alcohol, and overeating in general. *First, do no harm.* Instead of shifting your cells into antioxidant overdrive by adding a phytochemical like sulforaphane, why not try quieting the inflammation and oxidative stress by removing those prime suspects first?

The Truth Shall Set You Free

I fully support using medicines and supplements to treat the symptoms of existing disease when necessary, but food-first approaches that directly address root causes of disease whenever possible are much preferred. Placing your faith in superfood myths may only serve to enable you to continue eating and drinking things that are working against you. So, my advice to you is this: choose whole foods that you enjoy eating that nourish and energize your brain, and avoid foods that damage your cells and your metabolism. If you choose to eat chocolate or drink wine, be careful with quantities, be mindful of the risks, and do so with the understanding that these are indulgences, not healthy habits.

We have been conditioned for decades not only to give plant foods the benefit of the doubt, but to give plant foods more credit than they deserve. This *plant-biased* mindset makes it difficult for us to see things as they really are, enabling both the superfood movement and the “plant-based” diet movement to flourish in the absence of scientific evidence, to the detriment of our collective mental health.

CHAPTER 15

The Plant-Based Brain: Going Out on a Limb

To meet the 2050 challenges for quality protein and some of the most problematic micronutrients worldwide, animal source foods remain fundamental.

—Martin Cohen and Frédéric Leroy, “The Dark Side of Plant-Based Food”

Given widespread beliefs that plant foods fight disease and animal foods cause disease, it is not surprising that plant-based diets are increasingly viewed as the healthiest diets we can eat. Yet, in most cultures, for most of recorded history, diets containing a mixture of minimally processed animal and plant foods were the norm. Vegetarian diets were unusual (as were plant-free carnivore diets), and vegan diets would have been, since vitamin B12 supplementation wasn’t available prior to 1952.¹

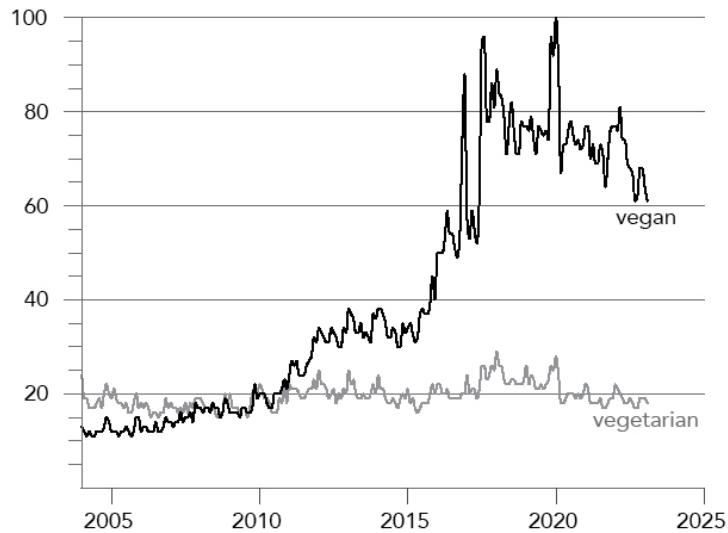
When we stand back and look objectively at the nutritional and biological differences between plant and animal foods, it is easy to appreciate that animal foods are nutritionally superior to plant foods and less likely to cause us harm. Therefore, making them the centerpiece of a mixed diet would seem to be the safest and most reliable way to keep ourselves healthy. Yet taking a plant-based approach to nutrition is increasingly recommended by influential health organizations such as the American Heart Association² and the World Health Organization³ as the best strategy for protecting human health.

The medical mainstreaming of plant-based dietary patterns is deeply troubling, as some of these patterns are sure to have serious unintended consequences for public mental health—I emphasize *some* of them, because the term “plant-based” means different things to different people.⁴ This ambiguity is problematic, especially when used in the context of medical

advice, where clarity is key. The term “plant-based” encompasses a wide variety of dietary patterns, from Mediterranean diets that allow all animal foods including lean red meat, to vegetarian diets that only allow dairy and/or eggs, to vegan diets that contain no animal foods whatsoever. From a biological perspective, there is a world of difference between vegetarian diets and vegan diets, so it is critical to make the distinction between them, which the term “plant-based” fails to do. For our purposes, I will use the word *vegetarian* to mean mixed diets that include dairy and/or eggs, the word *vegan* to mean diets that contain no animal foods at all, and the term *near-vegan* to describe any dietary pattern that is too low in animal foods to meet nutritional requirements without supplementation.

Of the 1.5 billion people in the world who eat a vegetarian diet, only 5 percent do so by choice; the rest don’t eat meat because they can’t afford it.⁵ This sliver of voluntary vegetarians includes people who have long avoided meat for religious or cultural reasons such as in India, where approximately 30 percent of the population follows a vegetarian diet.⁶ It also includes a growing segment of the population in Western countries, where secular vegetarianism is becoming more popular. Surveys suggest that 5 percent of adults in the United States, the UK, and Germany, and 8 percent of Canadians now identify as vegetarian.⁷ More than 10 percent of people living in Australia, New Zealand, Israel, and Sweden now identify as vegetarian or vegan.⁸

Vegan and near-vegan diets are capturing the hearts and minds of consumers concerned about public health, animal welfare, and the environment. As you can see in the graph below, Google search data reflects these trends. While interest in vegetarianism has remained steady since 2004, interest in vegan diets began rising in 2016.



Google Search Trends for "Vegan" and "Vegetarian"

RELATIVE NUMBER OF GOOGLE SEARCHES FOR “VEGAN” AND “VEGETARIAN” IN THE UNITED STATES BETWEEN JANUARY 2004 AND FEBRUARY 2023.

Google Trends indicates the relative popularity of the search terms as a ratio where 100 represents the peak popularity.

Data source: Google Trends (<https://www.google.com/trends>)

There are many reasons why people choose not to eat animal foods. Most college students I worked with who followed vegetarian or vegan diets told me that they chose to do so not for health reasons, but for compassionate reasons—because they cared about the health and well-being of animals and the planet. Since mainstream medical advice supports their choices by recommending vegetarian and vegan diets for optimal health, they felt unconflicted—better health was the icing on the cake, not something to be sacrificed for higher ideals. Unfortunately, good physical and mental health is precisely what is at stake, because the science behind plant-based diets says something very different than what we have been led to believe.

PLANT-BASED DIETS ARE NOT EVIDENCE-BASED DIETS

There is currently no scientific evidence available to help us understand how vegetarian and vegan diets affect psychiatric conditions. However,

there have been quite a few clinical trials demonstrating that vegan and vegetarian diets can indeed benefit other conditions like type 2 diabetes and cardiovascular disease. Since improving metabolic and vascular health could improve mental health, it is worth taking a closer look at this research.

While there are many studies to choose from, some of the best known and most highly cited work in this field has been conducted by physician researchers Dr. Dean Ornish, Dr. Caldwell Esselstyn, and Dr. Neal Barnard, all of whom have written popular books recommending the specific diet plans they have each studied. (You may also be familiar with Professor T. Colin Campbell's book *The China Study*, which argues that animal protein causes cancer, and that people should therefore eat a whole-foods vegan diet. Since the information in his book is based on nutritional epidemiology rather than human clinical trials, I will not analyze its findings here.)

Dr. Dean Ornish's 1990 Lifestyle Heart Trial demonstrated that a very low-fat vegetarian diet reduced the degree of stenosis (narrowing) in the coronary arteries of people with heart disease, leading to claims that his program reverses heart disease.⁹

This study was a one-year randomized controlled trial in which twenty-eight patients with coronary artery disease were prescribed a diet of grains, legumes, vegetables, fruit, egg whites, and non-fat dairy products. They were also told to avoid added fats, oils, and simple sugars, to limit alcohol and caffeine, exercise thirty minutes a day, practice daily stress management techniques, attend support groups, stop smoking, and take a B12 supplement. This group was compared to a control group of twenty patients who were not given any diet or lifestyle advice. After one year, the degree of stenosis in the Ornish diet group had decreased by 2.2 percent—a significant improvement compared to the control group, in which stenosis had worsened by 3.4 percent.

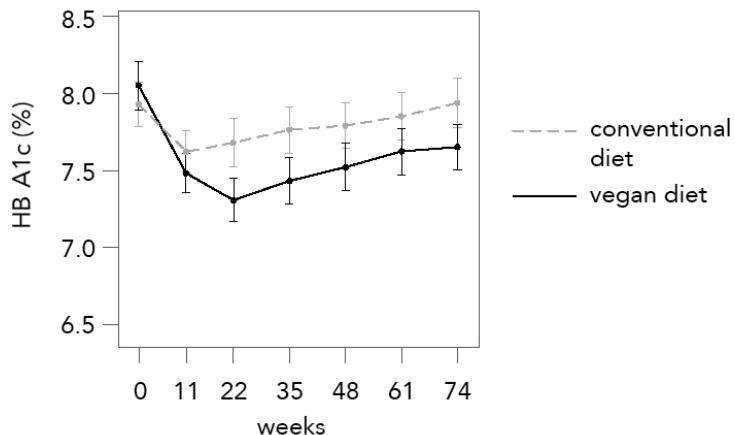
Dr. Caldwell Esselstyn is perhaps best known for demonstrating that people could arrest and reverse the progression of coronary artery disease with a very low-fat vegetarian diet. In his 1995 Arrest and Reverse Study, which lasted more than five years, twenty-two patients with severe triple-vessel coronary artery disease (more than 50 percent blockage) were prescribed a vegetarian diet of grains, legumes, lentils, vegetables, fruit, skim milk, and nonfat yogurt, that excluded oils and limited alcohol and

caffeine. Patients were also prescribed cholesterol-lowering medications and a daily multivitamin and attended regular support groups. Among the seven patients who completed the trial, there was a significant reduction in stenosis of 7 percent, on average.¹⁰

Dr. Neal Barnard has conducted numerous clinical trials of a vegan diet in a variety of health conditions including type 2 diabetes and obesity. The diet he uses in his research excludes dairy and eggs but is otherwise identical to the Ornish and Esselstyn diets. He recommends favoring low-glycemic foods and taking a B12 supplement but does not ask patients to exercise or make other lifestyle changes. Dr. Barnard claims his diet reverses type 2 diabetes because it lowers hemoglobin A1C (HbA1C)—a reflection of average blood sugars over the previous three months.

The internationally recognized definition of type 2 diabetes remission is the ability to maintain HbA1C below 6.5 percent for longer than three months without diabetes medication.¹¹ In Barnard's seventy-four-week RCT of ninety-nine patients, the forty-nine who followed the vegan diet did fare better than those following conventional dietary advice, but none lowered their HbA1C levels into the non-diabetes range.¹²

By contrast, low-carbohydrate diets have been shown in large studies to place type 2 diabetes into remission. When Indiana University obesity medicine specialist Dr. Sarah Hallberg and collaborators at Virta Health placed 349 patients on a low-carbohydrate diet for one year, average HbA1C levels fell from 7.6 to 6.3.¹³ British National Health Service physician Dr. David Unwin showed similar success with 186 patients on a low-carbohydrate diet; HbA1C levels fell from 7.9 to 6.4, with 51 percent of patients achieving remission over a nearly three-year period of observation.¹⁴ These were not randomized controlled trials, but they do indicate it is not necessary to remove animal foods from your diet or eat a low-fat diet to put type 2 diabetes into remission. Fat and protein don't raise blood sugar; carbohydrates do. The reductions in HbA1C that Dr. Barnard observed were likely related to the avoidance of refined carbohydrates, as he advised participants to "favor low-glycemic index foods, such as beans and green vegetables."



BARNARD STUDY RESULTS

Low-glycemic vegan diet modestly reduces hemoglobin A1C.

Neal D. Barnard et al., “A Low-Fat Vegan Diet and a Conventional Diabetes Diet in the Treatment of Type 2 Diabetes: A Randomized, Controlled, 74-wk Clinical Trial,” The American Journal of Clinical Nutrition 89, no. 5 (2009): 1588S-96S, <https://doi.org/10.3945/ajcn.2009.26736H>.

What the Ornish, Esselstyn, and Barnard studies tell us is that diet and lifestyle changes can make a positive difference in your cardiovascular and metabolic health, and that is an empowering message. Where the message gets muddled is that these studies are frequently pointed to as evidence that removing meat from the diet is what makes the difference. However, *none of these plans simply removes meat from the diet*. They also remove almost all of the fat (including vegetable oils), all of the refined carbohydrate, and all of the processed foods. In many cases, numerous other changes were also implemented such as exercise, smoking cessation, and cholesterol-lowering medication. It is therefore impossible to know whether the absence of meat had anything to do with the health benefits of any of these plans.

These studies are not exceptions; I am not aware of any human clinical trial of any health condition that removes animal foods without changing other key aspects of the diet. This means that **there is currently no scientific evidence that simply removing animal foods from the human diet brings any health benefits whatsoever**.

Until such studies are conducted, we are left to wonder: What if the health advantages of *whole-foods plant-based* diets have everything to do

with eating *whole foods* and nothing to do with eating *plant based*? In my thirteen years as a college psychiatrist, it was common to meet students who ate processed-foods vegan diets, but rare to meet students who ate whole-foods vegan diets (or whole-foods diets of any kind, for that matter). Food culture on most college campuses is a travesty. Other than avoiding animal foods, students choosing vegan diets ate the way most other students did—enormous sweetened coffee drinks, granola bars, sugary smoothies, pasta, muffins, chips, chocolate, energy drinks, and cookies all day long and well into the night. Combining the nutrient deficits of an under-supplemented, poorly planned vegan diet with the inflammation, oxidative stress, and high insulin levels that come with the standard American diet is a surefire prescription for brain damage and dysfunction. Despite faithfully avoiding all animal foods, none of these kind, talented, ambitious young people enjoyed good mental or physical health. Obesity, acne, tooth decay, type 2 diabetes, thinning hair, iron deficiency, chronic pain, depression, anxiety, ADHD, and overeating disorders were all too common among these young adults in the prime of their lives. Having studied this subject for more than fifteen years, and having worked with thousands of patients, one thing is clear: of all the reasons to choose a vegan diet, better mental health is not one of them.

BEYOND B12: NUTRITIONAL AND MENTAL HEALTH RISKS OF VEGAN DIETS

It is impossible to meet the brain's nutritional requirements through plant foods alone. All respectable health organizations are unanimous in recommending B12 supplementation for vegan diets, because all plant foods lack B12; in fact, strictly speaking, B12 is the only essential nutrient missing from all plant foods. However, the whole truth is that nutrient deficiency issues in vegan diets go well beyond B12. While nutrition authorities around the world disagree wildly about which nutrients other than B12 may require supplementation, most authorities, including those that strongly advocate for near-vegan and vegan diets, do acknowledge that additional risks exist.

In 2019, an international group of thirty-seven scientists led by Professor Walter Willett published an influential report titled, "Food in the

Anthropocene: The EAT-Lancet Commission on Healthy Diets from Sustainable Food Systems.”¹⁵ This commission’s task was to devise a strategy that could achieve an environmentally sustainable and optimally healthy diet for the world’s people by 2050. The group concluded that the best way to accomplish this goal would be to reduce the amount of animal food in the global human diet to as little as 0 grams per day. In their report, the authors note that strict vegan diets require B12 supplementation, but they also go on to write that their diet plan may not meet the iron and omega-3 fatty acid needs of pregnant women, nor the iron needs of teenage girls. They recommend animal foods for malnourished populations and recommend breastfeeding for children under two years of age (implying that their diet plan is not suitable for children under two). They acknowledge that animal proteins are of higher quality than most plant protein sources, and that high-quality protein is “particularly important for growth of infants and young children, and possibly in older people losing muscle mass later in life.”¹⁶ Unfortunately, as is too often the case when reading literature about vegan diets, identifying these important exceptions and precautions requires close reading, the parsing of convoluted rhetoric, and some special nutrition knowledge.

EAT-Lancet’s influential report is just one example of the common practice of recommending near-vegan and vegan diets while minimizing the nutritional risks. It is one thing for people to choose a vegan diet for their own personal reasons. It is quite another for medical professionals to recommend it for health reasons without explicitly calling attention to its health risks and giving clear, detailed advice about menu planning and supplementation. Just as with any other piece of medical advice, we physicians must present both the risks and the benefits to our patients so they can make an *informed* choice. To do otherwise is medically irresponsible and potentially dangerous.

In 2022, researchers in the Netherlands conducted a systematic review of dozens of nutrient measurement studies¹⁷ and found that vegans were more likely to be deficient in vitamin B12, vitamin D, zinc, EPA, DHA, and iodine (more than 90 percent of vegans had iodine deficiency). Vegans were also more likely to have low bone density, and vegan women were more likely to have iron deficiency.

The brain requires every essential nutrient to function properly, so if you

have a deficiency in any one of them, your mental health could suffer. How a nutrient deficiency will affect you depends on many factors, including how old you are when the nutrient deficiency begins, how severe the deficiency is, how long it lasts, and what other deficiencies and health problems you have. If a deficiency begins at an early age, persists for a long time, or is very severe, irreversible damage could occur.

Nutrient Deficiency	Mental Health Risks
Vitamin B12	Behavior change, psychosis, cognitive impairment 18
Iron	ADHD, 19 anxiety, depression, psychosis, sleep disorders 20
Zinc	ADHD, 21 depression, 22 psychosis 23
Iodine	Hypothyroidism, anxiety 24
DHA/EPA	ADHD, autism, mood disorders, schizophrenia, dementia 25

The relationship between most nutrients and most psychiatric disorders is still unclear, because without the option of conducting human clinical trials, the best we can do is to observe associations and guess about causation. The most important exception to this rule is B12 deficiency, where we commonly see clear reversal of severe psychiatric symptoms with supplementation.

Numerous case reports and studies find that vitamin B12 deficiency can lead to a wide variety of serious psychiatric issues, from depression and psychosis to delirium and dementia.[26](#) For example, when researchers in Pakistan examined one hundred young adults who had been eating a lacto-vegetarian diet (a vegetarian diet that includes dairy but not eggs) since childhood and compared them to one hundred young omnivores, they found that fifty-one of the vegetarians had B12 deficiency (compared to only three of the omnivores). To make matters worse, thirty-one of them had depression, eleven had psychosis, and seven had memory impairment; depression was more than twice as common and psychosis was more than three times as common among the vegetarians.[27](#) In an extreme example, a young mother who had gradually become deficient in B12 while following a vegan diet developed severe dementia to the point that she lost the ability

to care for herself and her child and could only utter one-word sentences. B12 deficiency can lead to permanent brain damage if left untreated for too long, but thankfully, with B12 injections she completely recovered in a matter of months.²⁸

If B12 is the only nutrient missing from all plant foods, why isn't B12 supplementation enough? Remember: just because a plant food contains a nutrient doesn't mean you can access it.

- Some plants contain antinutrients that interfere with nutrient absorption.
- Some plant nutrients must be converted into human-friendly forms before we can use them.
- Some of the nutrients that plants lack are conditionally essential.

Plant Nutrient Shortcomings

Plant Nutrient Limitation	Essential Nutrient
Not found in plants	Vitamin B12
Require conversion	DHA and EPA Vitamin K2 Vitamin A
Poor bioavailability	Iron Zinc Calcium
Limited plant food sources	Iodine Choline Selenium Lysine, methionine and glycine (amino acids)
Conditionally essential ²⁹ (required only if lysine and methionine are insufficient in the diet)	Carnitine (made from lysine and methionine) Taurine (made from methionine and cysteine) ³⁰

Unfortunately, there is little consensus among nutrition authorities about which, if any, of the above nutrients need to be supplemented on a vegan diet, because it *might* be possible *under certain circumstances, with careful planning*, to meet your requirements for them by eating plants alone. These nutritional gray areas open the door to the potential for serious nutrient deficiencies, especially for people who have higher nutrient needs, such as people with gastrointestinal conditions that affect nutrient absorption; people over 60; people who are recovering from illness, injury, or surgery; people with chronic illness; and people who don't have access to enough nutritious plant foods or quality supplements. All too often, these risks are overlooked, downplayed, or dismissed by nutrition authorities, creating confusion and controversy around supplementation requirements.

These important considerations are what I call the “ifs, ands, and buts” of vegan diets. My position is that a vegan diet, while not optimal, *might* be safe:

- IF you plan your meals primarily around nutritious whole foods
- AND you supplement wisely
- BUT not if you are pregnant, breastfeeding, or still growing

THE FIRST THOUSAND DAYS

One of the greatest gifts a mother can give a child is a big, beautiful, healthy brain. If we want to make a dent in our global mental health crisis, we don't just need more effective ways to treat mental illnesses, we need to find ways to prevent them from occurring in the first place. It is during the first thousand days of life—between conception and age two—that critical milestones in brain development occur. This is the single most important construction project of our lives, and every phase of the process is timed right down to the day. If precisely the right mix of nutrients isn't available precisely when needed, major problems can ensue, ranging from intellectual disabilities to serious birth defects to spontaneous miscarriage.

Advanced parental age, environmental pollutants, socioeconomic deprivation, and substance use can all cause brain development to go awry as well, but inadequate nutrition is the simplest (and arguably most

important) of these risk factors to address, because food provides the basic building blocks needed to grow a healthy brain. The good news is that if you have access to healthy foods and you have accurate information about what a healthy diet is, you can feed your baby the ingredients needed to lay the foundation for a lifetime of excellent mental health. But in order to succeed in this task, you must include animal foods in your diet.

Not only is it easier for new moms to nourish new brains if they include animal foods in their own diets, it is virtually impossible to meet baby's nutritional needs without them. The nutrients of most concern during pregnancy—meaning those required in the largest quantities and hardest to obtain from standard modern diets—are choline, vitamin D, DHA, EPA, folate, iodine, and iron. (Future mothers eating a vegan or vegetarian diet also need to add vitamin B12 and taurine to this list.) With the exception of folate, all of these micronutrients are either harder to find in plant foods than in commonly consumed animal foods or don't exist in plant foods at all, placing babies of vegan and vegetarian mothers at significant risk for nutritional deficiencies. For example, the World Health Organization and UNICEF recommend all infants receive breastmilk and only breastmilk for at least the first six months of life, and that they continue on breastmilk (in combination with other foods if desired) until at least age two. However, when researchers analyze breast milk from well-nourished mothers following vegan and vegetarian diets, they find it tends to be too low in B12, DHA, EPA, and taurine, so these must be supplemented.³¹

It is difficult enough to find adequate quantities of these irreplaceable animal-sourced nutrients in a typical omnivorous diet, let alone in near-vegan and vegan diets, which intentionally minimize or eliminate animal foods. Yet, despite these serious concerns, national guidelines in the United States, UK, Canada, Australia, and New Zealand all either explicitly support vegan diets for pregnant and breastfeeding women or do not directly address the issue, and those in support vary substantially in their supplementation recommendations.

Given how complicated an undertaking it can be to learn about, properly plan, adequately supplement, and medically monitor a vegan pregnancy, and given the increased nutritional requirements of developing infants, children, and teens, some national health organizations such as the German Nutrition Society; the European Society for Pediatric Gastroenterology,

Hepatology, and Nutrition; and the Royal Academy of Medicine in Belgium explicitly advise *against* vegan diets during pregnancy, breastfeeding, childhood, and adolescence.³² The Royal Academy of Medicine in Belgium issued a compelling position statement on this issue in 2018, writing in part:

It is not medically recommended and even prohibited to subject a child, in particular during periods of rapid growth, to a potentially destabilizing diet, justifying supplements and requiring frequent clinical and biological checks. This feeding concept... no longer resembles a conventional diet but a form of “treatment” that is unethical to impose on children.³³

Let's put our molecules in the context of the miracle of brain development so you can see how pivotal they are to the process and appreciate what could go wrong without them.

A Brain Is Born

During week three of pregnancy, before many women even realize they are pregnant, an unassuming little sheet of nerve cells quietly starts expanding and curling in on itself. By day twenty-seven, its edges have met and zipped themselves together to form a tube. The brain will begin bulging out of the top of this tube by week six, and the rest of the tube will become the spinal cord. Within three short weeks, the foundation for baby's entire central nervous system has been poured. This tight timeline depends on vitamin B12 and folate, which work together to make copies of new DNA molecules for cells that are busily multiplying to create these brand-new structures. If either of these vitamins is in short supply during the first month of pregnancy, the neural tube will be left unfinished, resulting in miscarriage or disfiguring *neural tube defects* such as spina bifida.³⁴ Women following vegan and vegetarian diets are not at higher risk for folate deficiency, but B12 deficiency is much more common in this group, and pregnancy further depletes reserves, so supplementation is critical.³⁵

Helping to direct the brain's development throughout the entire pregnancy is thyroid hormone, which is made with iodine. Iodine is so

crucial to normal brain development that severe deficiency (which was widespread before iodized salt programs) leads to serious developmental disabilities. Mild to moderate deficiency, which is still common even in Europe and the United States, can lead to lower IQ scores and subtle intellectual disabilities. Vegan diets increase risk for iodine deficiency because most plant foods are low in iodine.³⁶

As the brain rounds the corner into the third trimester, it enters a marathon of intensive brain building that continues until age two. Brain cells busily multiply to form new structures, migrate to new destinations, and myelinate new axons to establish secure communication pathways. This whirlwind of activity requires membranes—lots and lots of membranes. To prepare for its long growth spurt, the brain begins hoarding massive quantities of DHA and choline to insert into the membranes of every new cell it creates.

The developing baby soaks up 40 mg of DHA per day, more than ten times the amount we absorb as adults. New synapses, new myelin, and new mitochondria all need DHA, but this invaluable omega-3 fatty acid isn't just a membrane building block—it also serves as a signaling molecule to guide new neurons to their assigned positions within the cortex, and it has powerful anti-inflammatory and antioxidant properties that protect the vulnerable new brain from damage.³⁷

Remember: plant foods contain absolutely no DHA; the only omega-3 fatty acid they contain is ALA, which we struggle to convert into DHA. Even pregnant women—who are better at transforming ALA into DHA than anyone else—can't make enough DHA for their ravenous babies' brains. Studies consistently find that, compared to mothers who eat omnivorous diets, DHA levels are one-third to two-thirds lower in the umbilical cord blood, breast milk, and babies of vegetarian and vegan mothers.³⁸ These observations have led most experts to agree that ALA alone is not sufficient to meet the developing brain's requirements for DHA.

The long-term risks of early life DHA deficiency are potentially devastating. We know that infants born prematurely, who miss out on the full supply of DHA they are supposed to receive during the third trimester of pregnancy, grow up with poorly connected, under-myelinated brain cell networks. Researchers at the University of Cincinnati have observed that the brains of people with ADHD, mood disorders, and psychotic disorders

are similarly underdeveloped.³⁹ Like an artist who runs out of paint, if DHA falls short during the third trimester, the developing human brain must leave its most prized masterpiece—the cortex—unfinished, just as it must in babies born prematurely who haven’t had the luxury of completing a full term of pregnancy.

Rolling out new membranes also requires copious quantities of choline, which explains why fetal choline levels during the third trimester are nearly six times higher than typical adult levels. Without choline, the brain can’t make acetylcholine, a neurotransmitter central to learning and memory, nor *phosphatidylcholine*—one of the most vitally abundant molecules in our membranes. Choline also regulates genes that help direct brain development. A shortage of choline poses problems for the entire brain, but is particularly perilous for the hippocampus (the brain’s learning and memory center), which relies heavily on choline to form and function properly. Since choline wasn’t recognized as an essential nutrient until 1998, we understand very little about how choline deficiency during pregnancy plays out in children and adults, but emerging research suggests it may contribute to risk for autism, schizophrenia, and cognitive impairment.⁴⁰ Many plant foods are low in choline, so vegan diets increase risk for choline deficiency. Despite its critical importance, more than 90 percent of pregnant women do not get enough choline (regardless of dietary preferences), and most prenatal vitamins still do not contain choline.⁴¹

Brain building also demands energy, which means lots of electron transport chains busily churning out ATP. Iron deficiency during pregnancy can cause irreversible damage to the brain because these tiny generators can’t operate without iron. The energy-hungry hippocampus is particularly vulnerable to iron deficiency in pregnancy, which can lead to lifelong memory problems. Babies born to iron-deficient mothers show less complexity in their gray matter networks, have more trouble recognizing their mothers’ voices, and have a higher risk for ADHD.⁴² An analysis of over half a million children in Sweden found that children of mothers who were diagnosed with iron deficiency anemia in the first thirty weeks of pregnancy were two to three times more likely to have autism spectrum disorders and intellectual disabilities.⁴³ The form of iron found in plant foods is less bioavailable, so vegetarian and vegan women of reproductive age tend to have lower iron stored in their bodies.⁴⁴

And finally, just as insulin is a master regulator of growth, so is vitamin D. Vitamin D is a steroid hormone needed from day one of pregnancy to orchestrate the complex process of brain development, fight damaging oxidative stress, and ensure proper function of the brain's immune system. During the third trimester it is heavily involved in bone building, so Mom must supply baby with ever-increasing amounts of vitamin D as pregnancy progresses: baby's vitamin D levels are supposed to double in the first trimester and then double again approaching the third trimester. Plant foods don't contain vitamin D, and the form found in mushrooms and yeast (which are technically not plants) is vitamin D₂, which is less effective at raising and maintaining our vitamin D levels than vitamin D₃, the form found in animal foods.⁴⁵ Vitamin D deficiency is common in general, but vegans and vegetarians are even more likely to be deficient and have higher rates of osteoporosis.⁴⁶ While several nutrient deficiencies during pregnancy have been linked to a possible increased risk for autism, the evidence is strongest for vitamin D deficiency, probably because of vitamin D's critical role in directing brain development throughout pregnancy.⁴⁷

VITAMIN D, SUNSHINE, AND INSULIN RESISTANCE

Vitamin D doesn't have to come from the diet at all, because we can make it ourselves if we get enough sunshine. In fact, even though the brain's need for vitamin D remains high after delivery, baby's vitamin D levels drop sharply after birth, because after nine months in darkness, Mother Nature expects babies to be exposed to sunlight so they can start making their own vitamin D, but modern indoor lifestyles and fear of skin damage mean many babies spend less time in the sun. As little as thirty minutes of midday sunlight per week may be sufficient,⁴⁸ although those with darker skin or who live far from the equator may need longer sun exposure time.

Lack of sun exposure is just one of the reasons why so many moms and infants are deficient in vitamin D. Another major force we all have to contend with is insulin resistance, which is so tightly tied to vitamin D deficiency that we used to

think vitamin D deficiency caused insulin resistance, but we are beginning to see that the relationship may be the other way around: Insulin resistance could be a contributing cause of vitamin D deficiency, because insulin resistance interferes with our ability to *activate* and *respond* to vitamin D.⁴⁹

The scarcity of these precious molecules in our modern diet may help to explain why prenatal vitamins are recommended for every future mom, regardless of dietary preferences. Unfortunately, the quality of prenatal vitamins varies greatly, and most still don't contain everything you need, so attention to nutrition is still very important. Furthermore, as leading prenatal nutrition expert Lily Nichols RDN warns: "Sadly, if you follow conventional prenatal nutrition advice, you're almost guaranteed to be eating a nutrient-deficient diet."⁵⁰

Therefore, if you are planning a pregnancy, be sure to shore up your diet with nutrient-rich animal foods from at least six months prior to conception until you've finished breastfeeding. Why take the unnecessary risk of withholding nutrient-rich animal foods during the brain's most critical window of development? Given how little we still know about optimal nutrient requirements during pregnancy and what the long-term psychiatric risks of nutrient deficiencies are, it would be wise to err on the side of caution.

Complete coverage of these critical topics is well beyond the scope of this book, so regardless of your dietary preferences, if you are pregnant, breastfeeding, or planning a pregnancy, I highly recommend you read *Real Food for Pregnancy* by Lily Nichols for expert guidance about proper nutrition and supplementation. Her comprehensive book includes thoughtful, well-researched advice for omnivores and vegetarians alike.

Finally, if you are considering a vegetarian or vegan pregnancy, please also consult with a medical professional with deep knowledge and experience in this area for personalized advice about supplementation, dietary planning, and clinical monitoring well before conceiving.

HOW TO OPTIMIZE VEGETARIAN AND VEGAN DIETS

FOR BETTER BRAIN HEALTH

If you are a *nonpregnant* adult and you eat a vegan or vegetarian diet, I offer some suggestions below to help you optimize your diet for better mental health. Even if you follow these suggestions, you may still need to supplement, but since supplementation advice will vary depending on your food choices, health circumstances, and other factors, please consult with a health care professional with deep knowledge and experience in this area for personalized guidance about supplements.

Protein (Amino Acids): Focus on Eggs, Nuts, and Tofu

Many plant proteins do not contain optimal quantities and proportions of all nine essential amino acids, with lysine and methionine being hardest to find. However, you can still meet your amino acid needs on a vegetarian or vegan diet if you plan properly.

Vegetarians: Eat eggs regularly. If you don't eat eggs, dairy products will need to be your source for animal nutrients; just keep in mind that they can be more problematic (see [chapter 11](#)).

Vegans: Legumes, nuts, and seeds are your richest whole food sources of amino acids (see [chapter 12](#) for information about how to reduce toxins and antinutrients in legumes). Calcium-set tofu is a complete protein with excellent bioavailability and has the added benefits of being low in carbohydrate and high in calcium. Other complete protein sources are quinoa, buckwheat (a gluten-free grain), and quorn (a protein isolated from fungi). Avoid flours of all kinds, processed cereal products, and grains that contain gluten (wheat, barley, rye, and triticale). If you use plant milks, choose unsweetened varieties that are free of vegetable oils. Avoid processed meat substitutes, especially those made with vegetable oils, sugar, and other industrially processed ingredients.

Fats: Focus on Fatty Fruits and Nuts

Avoid "buttery spreads," vegetable oils, and trans fats. Obtain your healthy fats by eating whole plant foods whenever possible: avocados, olives, palm fruit, nuts, seeds, and coconut. Healthier added fats include olive oil, avocado oil, palm fruit oil, cocoa butter, sesame oil, and cold-pressed nut

oils.

Carbohydrates: Focus on Fruits and Vegetables

If you do not have insulin resistance yet, you can enjoy fruits and starchy vegetables in your diet. If you want to *keep* your metabolism healthy, choose low glycemic varieties (see [chapter 7](#)) and strictly avoid refined carbohydrates.

If you have insulin resistance or type 2 diabetes, I encourage you to consider a low-carbohydrate diet, in which case minimizing or even eliminating grains, starchy legumes, fruits and starchy vegetables would become important. It isn't difficult to construct a low-carbohydrate vegetarian diet, but low-carbohydrate vegan diets pose special challenges. Nevertheless, low-carbohydrate vegan diets are possible if you emphasize lower-carbohydrate protein sources such as soy, macadamias, almonds, and hemp seeds.

BETTER BRAIN HEALTH FOR EVERYONE

The next and final section of this book lays out the three dietary strategies I use most often in my clinical work, and the core of all three of them is animal foods. However, if including some animal foods in your diet isn't an option for you, the suggestions above will go a long way toward helping you better nourish, protect, and energize your brain. I would still encourage you to read the final chapters, as there are additional brain food rules worth knowing about, as well as important information about ketogenic diets and food sensitivities that apply to everyone, regardless of dietary preferences.

PART 4

HOPE IS ON THE MENU

CHAPTER 16

The Quiet Diet Approach

Now comes the time for a new beginning.

We've been fed the wrong information about nutrition for generations, which means that, if you're like most of us, you've been feeding your brain improperly all your life. The silver lining around this cloud is that you have no idea how much better you could feel if you ate a brain-healthy diet. Get curious! You owe it to yourself to discover what's possible. Your outlook on life could change in ways you wouldn't have imagined.

BRAIN FOOD RULES

Which dietary changes are most worth making, and why? Let's return to our touchstones of *nourish*, *protect*, and *energize* and create a fresh new set of brain food rules grounded in biology instead of ideology, questionnaire-based guesswork, or wishful thinking.

A brain-healthy diet must:

Nourish the brain by providing all essential nutrients.

- Include animal foods because they are nutrient-rich.
- Avoid plant foods high in antinutrients, especially grains and legumes.

Protect the brain by excluding foods that contain damaging ingredients.

- Follow whole-foods principles.
- Avoid refined carbohydrates, vegetable oils, and alcohol because they cause inflammation and oxidative stress.

- Avoid high-toxin plant foods such as cassava (toxic to mitochondria and the thyroid), flaxseed (potentially toxic to mitochondria), and most nightshades (toxic to the nervous system).
- Consider eliminating all dairy products.
- Listen to your body. Limit any food that causes you physical or psychological side effects such as digestive distress, insomnia, fatigue, or brain fog, as symptoms such as these may indicate inflammation.

Energize the brain in ways that protect brain metabolism over the life span by keeping blood glucose and insulin levels in a healthy range.

- Avoid refined carbohydrates.
- Avoid dairy protein powders.
- Customize carbohydrate quantity to achieve healthy glucose and insulin levels.

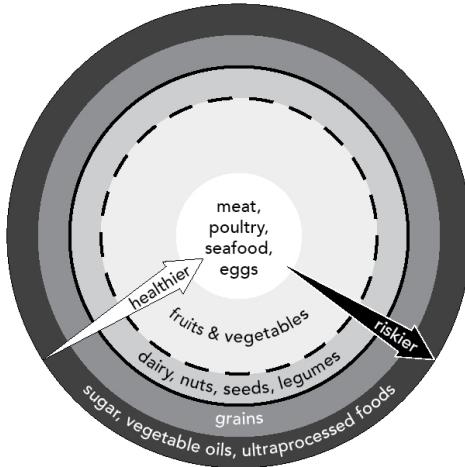
Practice Whole-Food Principles

Whole foods don't require nutrition labels, so the best nutrition label is no nutrition label at all. My definition of a whole food:

1. Whole foods are single-ingredient plant or animal foods that can be found in nature (an egg, a peach, a fish)
2. Whole foods are perishable (good food goes bad)
3. Whole foods require little to no processing to be safely edible (peeling, chopping, and cooking are fine)

If we follow these brain food rules to their logical conclusion, we don't arrive at a Mediterranean diet, because Mediterranean diets require grains, legumes, and dairy products, allow substantial quantities of refined carbohydrates, and encourage alcohol. We don't arrive at a vegan diet, because vegan diets lack animal foods and therefore can't provide all essential nutrients without relying on legumes, fortified processed foods, and supplements. The broadest dietary pattern that comes closest to

satisfying these brain food rules is the *paleo diet*, because it emphasizes animal foods and excludes grains, legumes, dairy, refined carbohydrates, alcohol, and modern processed foods. If you have insulin resistance or type 2 diabetes, you may need to follow a lower-carbohydrate version of the paleo diet or even transition to a *ketogenic diet*. If you have significant damage to your gut health or immune system, you may have lost the ability to tolerate a wide variety of plant foods, in which case you may even benefit from a plant-free *carnivore diet*, at least as a temporary discovery strategy.



CORE PRINCIPLES OF A BRAIN-HEALTHY DIET

The core of your diet should be non-dairy animal foods supplemented with fruits and vegetables as tolerated. Dairy, nuts, seeds, and legumes offer nutritional value but are risky. Grains, sugar, vegetable oils, and ultraprocessed foods are best avoided entirely.

Suzanne Smith

If you would like to try a standard paleo, ketogenic, or carnivore diet, I've included some of my favorite resources in appendix B to help you make the most of those plans. However, if you've already tried those approaches and they haven't brought you enough relief, or if you're new to the world of special diets and want to discover the dietary pattern that will work best for you as efficiently as possible, my Quiet Diet plans are here to help take the guesswork out of your journey.

THE QUIET DIET DIFFERENCE

While standard paleo, ketogenic, and carnivore diets go a long way toward fulfilling our brain food rules, they each fall short in ways that may make it harder for you to achieve your personal best, which is why I created Quiet Paleo, Quiet Keto, and Quiet Carnivore alternatives for you to explore. I call these plans “quiet” because they are uniquely modified to be quieter on your metabolism, gut, thyroid, immune system, nervous system, and mitochondria, allowing them to deliver benefits beyond standard paleo, ketogenic, and carnivore diets.

What makes Quiet Diet plans different from standard paleo, keto, and carnivore plans is that all three Quiet Diets:

- lower glucose and insulin levels
- reduce inflammation and oxidative stress
- minimize plant toxins and antinutrients
- eliminate common food sensitivity culprits
- emphasize gut-friendly foods that are easier to digest

The Quiet Diet approach can be particularly helpful if you are struggling with:

- irritable bowel syndrome (IBS)
- chronic pain or fibromyalgia
- migraine headaches
- chronic fatigue
- food addiction
- stubborn or mysterious mental or physical health issues
- food sensitivities or chemical sensitivities

Food intolerances are increasingly common, affecting up to one in five people in industrialized countries.¹ The cells lining the insides of your intestines work together with your microbiome and your immune system to decide which food molecules should be absorbed and which ones should be rejected. If any element of this sophisticated system becomes compromised, you could develop abnormal reactions to foods. There are no definitive

explanations as to why so many of us are losing our ability to tolerate a wide variety of foods, but several compelling theories exist. One is the concept of *toxicant-induced loss of tolerance* or TILT. The idea behind TILT is that exposure to environmental toxins such as pesticides, petrochemical solvents and fumes, and microplastics²—all of which have become prevalent only since World War II—weakens the immune system, making us exquisitely sensitive to foods and chemicals we used to be able to tolerate. Other potential culprits include antibiotics that deplete our microbiome,³ food additives such as emulsifiers (polysorbate 80, lecithin, xantham gum, carageenan, and many others) that cause intestinal inflammation,⁴ and *endocrine disruptors*—substances that upset hormonal balance, function, and rhythm such as plastics, soy phytoestrogens (plant estrogens), and pesticides.⁵

Regardless of the original cause(s), growing evidence suggests that people with food allergies and intolerances are more likely to have increased intestinal permeability, aka “leaky gut”—gaps in the tight junctions between intestinal cells that are supposed to prevent undigested food particles and other unwanted substances from crossing into the bloodstream.⁶ If these sneak into the circulation where they don’t belong, your immune system could view them as foreign intruders and mount an inflammatory reaction against them.

In the case of true food allergies, even a tiny quantity of food can trigger the body to immediately release a massive amount of *histamine*—a powerful neurotransmitter that can cause hives, throat swelling, wheezing, and other familiar allergy symptoms that can even be life-threatening in some cases. Food sensitivity symptoms, on the other hand, are far less dramatic, less specific, and more diverse. Food sensitivity reactions may include any number of frustrating symptoms such as acne, asthma, indigestion, fatigue, migraine, joint pain, bloating, stomachache, ankle swelling, brain fog, anxiety, insomnia, and depression. Symptoms can take hours to days to appear, and their severity depends on the quantity of food you eat, so small amounts of food may not bother you unless you eat it very frequently.⁷

Because symptoms can be so variable and so vague, many people with food sensitivities don’t realize they have them. If you suffer from environmental or chemical sensitivities, or a diagnostically perplexing

“mystery syndrome” such as fibromyalgia, chronic fatigue syndrome, or irritable bowel syndrome, you could very well have unrecognized food sensitivities. Unfortunately, despite what home test kit manufacturers claim, there are no reliable tests for food sensitivities,⁸ so the only way to identify a sensitivity is by eliminating the suspected ingredient from your diet for a while as an experiment; in other words, *when in doubt, cut it out*. You could remove one food at a time and then reintroduce it after a few weeks, but this elimination diet strategy can be tedious, especially since it is unusual to have just one food sensitivity. Given that these issues now affect so many people, all three quiet diets are free of the most common food sensitivity culprits to spare you the frustration of the trial-and-error elimination process.

Standard and Quiet Paleo Diets

The paleo dietary pattern was first popularized by Colorado State University professor Dr. Loren Cordain. “Paleo” is short for paleolithic, which means “Stone Age.” A paleo diet attempts to mirror the way our hunter-gatherer ancestors ate for nearly two million years, prior to the dawn of monocrop agriculture and the domestication of animals. Simply put, the paleo diet is the original human diet. As Professor Cordain wrote in his book *The Paleo Diet*: “Our genes are well adapted to a world in which all the food eaten daily had to be hunted, fished, or gathered from the natural environment.”⁹ The paleo diet permits meat, seafood, poultry, eggs, fruits, vegetables, nuts, and seeds—whole plant and animal foods that require little to no processing to be safely edible. Of course, it’s impossible to eat *exactly* the way our prehistoric ancestors did because the quality of our plant and animal foods has changed considerably since then. Following a paleo diet is less about eating exactly the same foods our ancient relatives would have eaten, and more about *avoiding* the foods they wouldn’t have eaten: grains, legumes, dairy products, industrially processed food products, refined carbohydrates, vegetable oils, alcohol, and added sugars.

Paleo diets haven’t been studied as much as Mediterranean or ketogenic diets have, but the handful of quality studies that do exist indicate modest metabolic health benefits in comparison to several other dietary patterns.¹⁰ Paleo diets usually do not require special medical supervision and are

appropriate for people of all ages. I am not a child psychiatrist, but when parents ask me what they should feed their children for better mental health, I recommend beginning with a paleo eating pattern.

While the standard paleo diet is a wonderful starting point for any brain-healthy diet, it may not sit well with you if you have gut health issues, food intolerances, or autoimmune diseases because it permits nuts and seeds, as well as the entire rainbow of fruits and vegetables, several of which can be problematic. The other potential drawback of standard paleo is that it can be too high in carbohydrate for the majority of us who now have insulin resistance, because it allows unlimited quantities of fruits and starchy vegetables. I created Quiet Paleo to address both of these potential issues.

A unique feature of Quiet Paleo is that it limits fruits and vegetables to my signature list of kinder, gentler plant foods that are easier to digest, lower in toxins and antinutrients, and have a lower glycemic index. For example, Quiet Paleo excludes cassava, flaxseed, and all nightshades (except carefully prepared potatoes) because they contain neurotoxins. While standard paleo plans place no ceiling on carbohydrate, Quiet Paleo limits total daily carbohydrate from low glycemic fruits and vegetables to about 90 grams per day to support lower, more stable glucose and insulin levels. If you find that your blood sugar and insulin levels still run too high on Quiet Paleo, I recommend you consider transitioning to a ketogenic diet.

Standard and Quiet Ketogenic Diets

The ketogenic diet isn't a dietary pattern that comes with a food list—it is a metabolic plan that comes with macronutrient rules: low carbohydrate, moderate protein, high fat. This means you can eat any foods you wish so long as you adhere to those rules and reach therapeutic ketosis. It also means that ketogenic diets can vary tremendously in quality depending on your food choices. In other words, all ketogenic diets energize your brain because they shift you into ketosis, but not all of them will properly nourish or protect your brain.

Most ketogenic plans include rich dairy products such as heavy cream and butter because these are delicious and easy ways to meet the high fat requirement, but dairy is also a common cause of inflammation and can raise appetite and insulin levels, resulting in unwanted weight gain. Many

ketogenic diets also rely on risky processed staples such as salad dressings made with vegetable oils, or “keto-friendly” treats like cookies made with nut flours, sweeteners, and whey protein. I understand the appeal of these items, but keep in mind that optimal brain health isn’t just about ketones. For example, if you’re following a ketogenic diet that falls short on nutrients or contains inflammatory ingredients, you may not experience much in the way of mental health benefits. These common shortcomings of most ketogenic diets led me to create the Quiet Keto plan.

The difference between Quiet Keto and other ketogenic diets is that Quiet Keto is based on the Quiet Paleo food list, so it excludes dairy, ultraprocessed ingredients, alcohol, and the most irritating plant foods. In short, Quiet Keto is simply the Quiet Paleo plan minus the higher-carbohydrate fruits and vegetables, giving you the best of both worlds. Like all ketogenic diets, Quiet Keto is metabolically quiet because it lowers glucose and insulin levels beautifully, but by focusing on low-toxicity whole plant and animal foods, it is safer and more nutritious than many other ketogenic diets, and therefore more likely to improve your overall health and sense of well-being. However, if you have compromised gut health or immune health, or if you suffer from multiple food sensitivity syndromes, Quiet Keto may still contain more plant foods than you can comfortably tolerate. Therefore, if you still don’t experience the results you are hoping for on Quiet Keto, you may want to consider trying a carnivore diet.

Standard and Quiet Carnivore Diets

The term “carnivore diet” has come to mean different things to different people, but the general philosophy behind this approach is that the fewer plants we eat, the healthier we will be, so carnivore diets are either extremely low in plant foods or contain no plant foods at all. Carnivore diets are naturally ultra-low in carbohydrate and relatively high in fat, so most carnivore diets are also ketogenic diets. Carnivore diets range from narrow beef-only plans to broad plans that allow all animal foods including dairy and eggs. Some versions of the carnivore diet even include spices, coffee, and alcohol, all of which come from plants. Quiet Carnivore is completely plant-free. Its very simple menu allows meat, seafood, poultry,

plain broths, and salt, but excludes dairy, eggs, and processed meats, because these are the animal foods most likely to bother sensitive individuals.

CHART YOUR COURSE

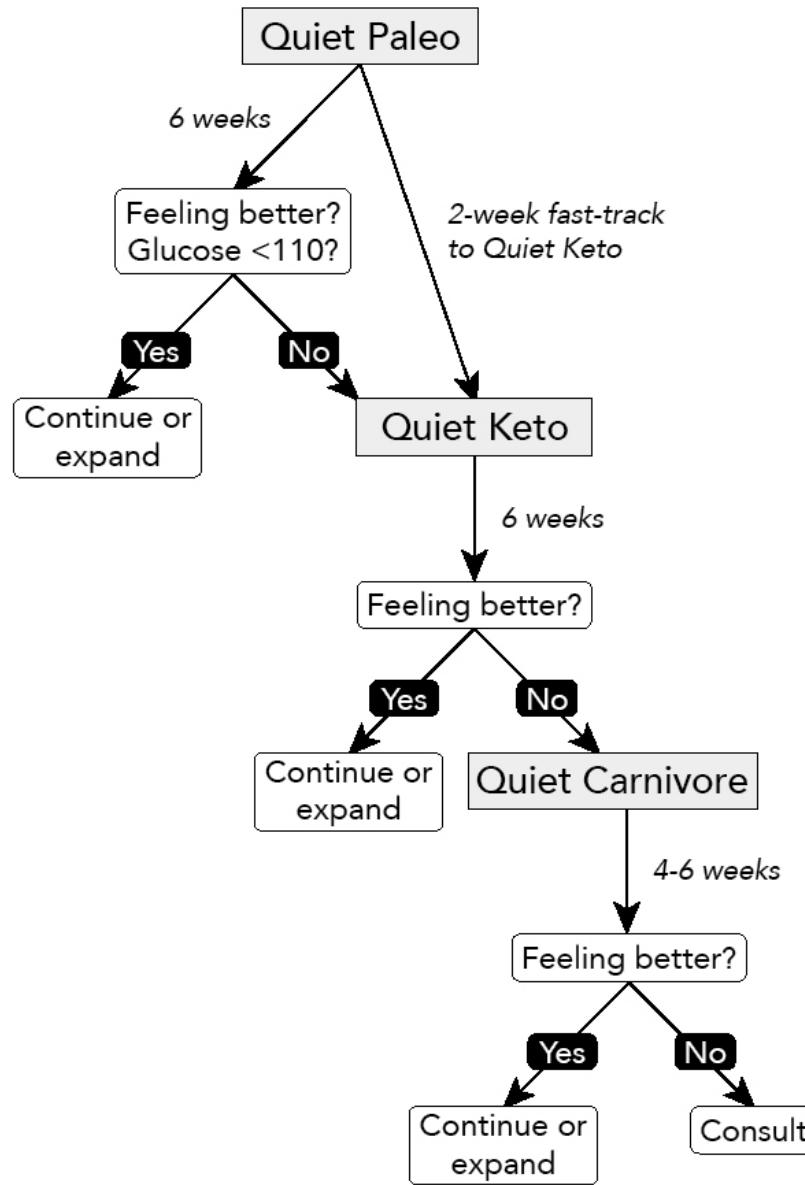
The most efficient way to discover the diet that will work best for you is by following the Quiet Diet roadmap below, which begins with Quiet Paleo; this is the strategy I recommend to most people. However, if switching immediately to Quiet Paleo sounds overwhelming, simply take whatever diet you are eating right now and make one healthy change at a time. It will probably take longer to achieve the results you're hoping for, but if a stepwise approach is easier for you, you're more likely to be successful in the long-term. Below are the most impactful single steps you can try:

- No added sugars
- No liquid calories (for example: no smoothies, sugar-sweetened beverages, milks, cream, or coffee lighteners)
- No grains
- No alcohol
- No snacking between meals
- No fast food
- No vegetable oils
- Fasting for sixteen hours every night

Recognize that if you choose the one-step-at-a-time approach, even though every step you take is good for your health, any one of these alone may not be sufficient for you to notice significant improvements in how you feel. Be patient and keep moving forward at your own pace. Continue to make healthy changes until you can really feel a difference.

THE QUIET DIET ROADMAP

The goal of the Quiet Diet process is to find the *least restrictive and most enjoyable* diet that will support your good mental and physical health. The roadmap starts with Quiet Paleo and then may or may not lead you on to Quiet Keto or Quiet Carnivore, depending on your needs. These special elimination diets are designed to help you figure out as quickly as possible whether your mental (and physical) health symptoms are rooted in food or metabolism issues without having to go through the frustrating trial-and-error process of removing one potential food culprit at a time. If you feel better on one of these plans, you can then try expanding your diet to discover where your safe outer limits are.



Everyone Start Here: Quiet Paleo (2 to 6 weeks)

Goals:

- To improve the overall nutritional quality of your diet
- To reduce inflammation by focusing on foods that are gentler to your gut, brain, and immune system

- To lower and stabilize your glucose and insulin levels, but without going into ketosis
- To serve as a two-week metabolic stepping-stone to Quiet Keto*

Follow Quiet Paleo for at least six weeks, then reassess your metabolism, your mental health, and your physical health. At that point, if your blood sugar is stable and in a healthy range, and you've noticed improvements in your physical and mental health, you can either continue Quiet Paleo long-term, or you can try expanding your food choices to see if you can tolerate a more varied paleo diet. However, if your blood sugar is unstable or runs too high on Quiet Paleo, or if you still aren't feeling as well as you'd like, then I would encourage you to consider switching to Quiet Keto.

*Even if you already know you'd like to try a ketogenic or carnivore diet, you should follow Quiet Paleo for at least two weeks first. This is very important, because diving headfirst from a standard diet that may contain hundreds of grams of carbohydrate per day into a ketogenic or carnivore diet that contains 20 grams of carbohydrate per day or less is a huge shock to the system that can temporarily cause unnecessary misery and could even be dangerous, particularly if you are taking certain medications or have certain pre-existing medical conditions (see [chapter 18](#)). Since Quiet Paleo starts at 90 grams of carbohydrate, it serves as a metabolic stepping stone that eases your glucose and insulin levels down more gradually, which will make transitioning to a ketogenic or carnivore diet much more comfortable.

Step 2. Quiet Keto (6 to 12 weeks)

Goals:

- To bring glucose and insulin levels down low enough for you to *enter* ketosis, and long enough for you to *adapt* to ketosis.
- To explore how being in ketosis affects your mental health.

After six weeks on Quiet Keto, reassess your metabolic, physical, and mental health. If you're satisfied with your results, you can either continue

Quiet Keto long-term or try gradually expanding your food choices as described below to see if you can tolerate a more varied whole-foods ketogenic diet while still maintaining the benefits of Quiet Keto. If Quiet Keto hasn't helped you enough (or at all), you may have multiple food intolerances, in which case you could try Quiet Carnivore.

Step 3. Quiet Carnivore (4 to 6 weeks)

Carnivore diets are ketogenic diets, so please read [chapter 18](#) before you begin to make sure a ketogenic diet is right for you. If you're fast-tracking from Quiet Paleo, I recommend following Quiet Carnivore for six weeks. If you're coming to Quiet Carnivore from Quiet Keto, you will already have been in ketosis for quite some time, so four weeks should be long enough to get a feel for the potential benefits of this plant-free protocol. Reassess your metabolic and mental health at either the four-week or six-week mark, as the case may be. If you are doing well, you can either continue Quiet Carnivore, or experiment with expanding your food choices.

HOW TO EXPAND YOUR DIET

If you feel better on Quiet Paleo, Quiet Keto, or Quiet Carnivore, but you're curious to see if you can tolerate a wider variety of foods, try reintroducing one new food type at a time to find your safe outer limit. For example, if you do well on Quiet Paleo but miss nuts, try eating some nuts every day for seven days to see how you feel. If that goes well, you can move on to the next curiosity experiment when you feel ready. You can dabble in cured meats, greater quantities of cruciferous vegetables, seeds, or a wider variety of spices, in any order you like. Keep a food and symptom journal to track your reactions. Identifying foods that make you feel worse doesn't necessarily mean you have to cut them out of your life forever; it's simply information that gives you more control over how you feel from day to day.

If you are blessed with a healthy metabolism and a robust constitution, you may be able to tolerate a wider variety of plant and animal foods, so when you're expanding your diet, where should you draw the line?

I recommend that all human beings draw the line at what we'll call ***brain-healthy paleo***: Enjoy meat, seafood, poultry, eggs, fruits, vegetables

(excluding cassava and all nightshades except carefully prepared potatoes), nuts, seeds (except perhaps flaxseeds), herbs, and spices. Continue to periodically monitor your after-meal blood sugar, fasting insulin, and fasting triglycerides. If any of these rise out of the healthy range, reduce your carbohydrate intake until you find your sweet spot.

Take care when expanding your diet to reintroduce foods with patience and with purpose so that you can more accurately and honestly assess how they make you feel. When relaxing a diet plan, it is easy to fall back into old habits and lose touch with your goals.

WHAT IF THE QUIET DIET PROGRAM DOESN'T WORK?

If none of these plans helps you, then your symptoms probably aren't diet related. However, I've worked with hundreds of patients, and can count on one hand the number of people who haven't experienced at least partial benefit from improving the nutritional and metabolic quality of their diet using these principles, so chances are excellent that you will benefit, too. If you don't improve enough or don't improve at all, I would still recommend that you continue to honor brain-healthy diet principles by following a paleo diet customized to your carbohydrate tolerance so that your glucose and insulin levels stay in a healthy range. This way, you will protect yourself from developing common diet-related mental and physical health problems in the future.

CHAPTER 17

Quiet Paleo

We've established that the first step toward changing your mind is to change your diet to a personalized paleo pattern, and this chapter will show you exactly how to do that. But before you begin, it's important to prepare yourself for the journey both medically and psychologically by taking stock of your mental and physical health, clarifying your goals, and setting yourself up for success.

MEASURE YOUR MENTAL HEALTH

In appendix A you'll find links to simple questionnaires to assess yourself for ADHD, depression, mania, anxiety, OCD, food addiction, eating disorders, and cognitive impairment. Choose the questionnaires that apply to your situation, fill them out, and tuck them away. (If you're not sure which ones to choose, fill them all out.) Six weeks after you change your diet, *without looking at your original questionnaires first*, fill out a fresh set of questionnaires, and compare them to your first set. Most people are pleasantly surprised to see how many things have improved in such a short period of time.

MEASURE YOUR METABOLIC HEALTH

Test yourself for signs of insulin resistance (see the list of insulin resistance tests [here](#)). You don't need to do every test on the list—just choose the ones that are easiest for you or your doctor to obtain. Here are the metabolic tests I recommend for everyone.

- Free and easy: waist-to-height ratio, blood pressure

- Inexpensive blood tests: fasting lipid panel (aka “cholesterol test”—you’ll need this to calculate your triglyceride-to-HDL ratio), fasting total insulin, fasting glucose, and hemoglobin A1C

If your clinician is hesitant to order these tests for you, you can order tests yourself through private direct-to-consumer laboratory services. Self-ordered tests aren’t covered by health insurance, but each of the tests listed above costs \$30 or less even without insurance coverage. If you live in the United States and have a Labcorp testing location near you, you can save money and support a good cause by using OwnYourLabs.com, a service established by cholesterol scientist Dave Feldman. A portion of the proceeds go to the Citizen Science Foundation.

If your results show many areas that need improvement, don’t be discouraged. Many markers of insulin resistance, such as high glucose, insulin, and triglyceride levels can improve substantially in just a few weeks using the dietary strategies in these chapters. Accept where you are right now without judgment and trust the process.

In addition to laboratory tests, I recommend checking your own glucose levels at home for at least a week before changing your diet and then for at least the first ten days of any dietary change. Revisit [chapter 7](#) for detailed information about home glucose monitoring and healthy glucose ranges. As a reminder, one way to measure your blood sugar is with a fingerstick blood glucose meter (many choices exist and they’re inexpensive). If you think you’ll ultimately progress to a ketogenic diet, I recommend purchasing one meter that measures both glucose and ketones (I use the Keto-Mojo GK+ blood glucose and ketone meter).

The best way to follow your glucose levels is with a continuous glucose monitor (CGM). CGMs are more expensive than fingerstick meters, but they’re painless and give you more information. CGM models, features, and prices are constantly evolving, so research current options before you buy or ask your health care provider for recommendations.

CONSIDER ADDITIONAL LABORATORY TESTS

Since insulin resistance isn’t the only reversible root cause of mental health

problems, I also recommend several additional laboratory tests to look for other common culprits including inflammation, certain nutrient deficiencies such as B12 and iron deficiency, and certain autoimmune conditions such as celiac disease and thyroid disease. Most people don't need extensive, specialized, or expensive tests—a few simple, carefully selected tests will usually suffice. You'll find a list of the laboratory tests I find most useful in appendix A. For personalized testing recommendations, consult your health care provider.

CONSIDER FOOD SENSITIVITIES

Since there is no laboratory test capable of diagnosing food sensitivities, you could have them without knowing it. As discussed, food sensitivities cause a variety of perplexing symptoms that may come and go; review the Body of Evidence checklist below to see how many of the most common symptoms you identify with. If you suspect you have food sensitivities, keep a food and symptom journal to look for connections between what you eat and how you feel.

Your Body of Evidence: Food Intolerance Symptom Checklist

If you have repeatedly experienced any of the following symptoms, place a checkmark in the box next to those symptoms and notice whether any of them improve when you follow Quiet Diet principles.

- Bloating
- Fatigue
- Headaches
- Aches and pains
- Brain fog
- Dark circles under your eyes
- Acne
- Itching
- Extremely dry skin
- Burning sensations in feet or hands

- Stomach pain
- Heartburn
- Sinus congestion
- Sneezing
- Flushed skin
- Racing or pounding heart
- Fluid retention
- Wheezing/coughing
- Constipation/diarrhea
- Gas/flatulence
- Nausea
- Plantar fasciitis (painful soles)
- Reflux
- Eczema

WHAT'S YOUR WHY?

Before you change your diet, take time to reflect and identify your personal motivations. Why do you feel a change is needed? Why now?

Describe in as much detail as you can how you have been feeling lately, both emotionally and physically, and then make a list of the mental health and physical health symptoms you hope to improve by changing your diet. Making a voice or video recording that you can listen to again six weeks after you've changed your diet can be especially powerful because it captures your tone, energy, and attitude in ways that writing can't.

Next, write down or record how you wish you could feel, and be as specific as you can. Divide your wish list into short-term goals (such as "I wish I felt more energetic") and long-term goals (such as "I wish I could stop my antidepressant"). Focus on the short-term goals for now.

UK-based clinical psychologist Dr. Jen Unwin and her husband Dr. David Unwin, a general practitioner who specializes in using low-carbohydrate diets to treat obesity and type 2 diabetes, created a simple, four-step process called GRIN to help people turn health wishes into realities.¹

G is for Goals. Choose up to three short-term personal goals. Not your

family's goals, not your doctor's goals—*your* goals. What difference would achieving these goals make in your life? How do you imagine your life would change? Be as specific as you can in envisioning your preferred future. For example, one patient told me she wanted to feel less irritable in hopes that her grandchildren would be more interested in spending time with her.

R is for Resources. What resources can you draw upon to help you achieve your goals? We've all accomplished difficult things before—what other challenges have you faced in your life, and how did you manage to conquer them? Make a list of your strengths. Not feeling positive about yourself lately? Ask others what strengths they see in you.

I is for Increments. If overhauling your diet all at once feels overwhelming, start small. Identify one change you feel ready to make, and once you've accomplished that one, set your sights on the next one. If you need ideas, there is a list of healthy changes in the previous chapter.

N is for Noticing. As you try changing your habits, notice your successes, no matter how small they may seem. At the end of each day, ask yourself: "What went well today?" None of us progresses toward change in a straight line. Mistakes are a normal part of the learning process. Focus on the positive and celebrate every little victory.

DON'T GO IT ALONE

Contact your medical and mental health care providers to let them know you want to try a new way of eating. If they are supportive of the idea, they can help by discussing your health goals, ordering lab tests, meeting with you periodically to monitor your progress, and making any necessary medication adjustments that may be needed along the way. Medical support is particularly important if you decide to try a low-carbohydrate diet of any kind (see [chapter 18](#)), whereas changing to a Quiet Paleo plan containing about 90 grams of carbohydrate per day is medically safe for most people. However, if your current diet contains hundreds of grams of refined carbohydrate per day and you are in fragile health, or taking medication for high blood pressure, high blood sugar, or heart disease, please do consult with your health care provider. A substantial drop in your carbohydrate intake may affect your blood sugar or blood pressure and may require that

your medication dosages be lowered.

Identify sources of social support such as family members, friends, or coworkers who can cheer you on or even join you on the journey for their own health reasons. Online and in-person support groups can greatly increase your chances of success.

If you need help finding medical or emotional support, see appendix B.

QUIET PALEO FOOD LIST

Meat, Seafood, Poultry, and Eggs

- All fresh or freshly frozen meat, seafood, poultry, and eggs are permitted. Meat, seafood, and poultry must be free of starchy coatings, thickened gravies, sweetened sauces, sweetened marinades, and sugary rubs.
- Minimize aged and processed meats such as cold cuts, bacon, aged beef, jerky, cured sausages, and smoked salmon as they can be high in histamine (see [chapter 19](#)).

Fats

- Non-dairy animal fats of all kinds: tallow, duck fat, goose fat, chicken fat, lard, etc.
- Fruit oils: extra virgin olive oil, unrefined avocado oil, unrefined palm fruit oil.

Vegetables

- Lettuces: all true lettuces are permitted, including iceberg, romaine, Bibb, Boston, green leaf, red leaf, oak leaf, Batavia, and butterhead. Avoid non-lettuce salad greens such as raw spinach leaves and raw cruciferous greens such as arugula, kale, raw cabbage, radicchio, mustard greens, and watercress.
- Endive
- Carrots*
- Mushrooms

- Globe artichokes
- Sunchokes (aka Jerusalem artichokes)
- Spinach (cooked)
- Celery
- Celery root (cooked)
- Parsnip* (cooked)
- Beets* (cooked)—but no beet greens
- Asparagus (cooked)
- Fennel
- Jicama
- Sweet potatoes*/yams* (cooked)
- Water chestnuts

*These vegetables have a higher glycemic index and may cause blood sugar spikes, especially if roasted or baked (boiling and stir-frying are somewhat better).

Fruits (remember: any food containing seeds is technically a fruit)

- All whole fresh or frozen fruits are permitted except goji berries (nightshades), garden huckleberries (nightshades), and star fruit (astronomically high in oxalates).
- Cucumbers
- Avocado
- Olives
- Squash: all varieties of squash are permitted including zucchini, yellow squash, pumpkin, delicata squash, spaghetti squash, butternut, buttercup, acorn, Hubbard, pattypan, etc.
- Limit tropical fruits and other high glycemic fruits to avoid blood glucose spikes. Watermelon, pineapple, banana, mango, grapes, and pears tend to raise blood glucose more than other fruits.
- Avoid dried fruit. Drying concentrates the sugars and removes the water, making dried fruit too easy to overeat. Many contain sulfites or other preservatives, and some are sweetened, such as blueberries and

cranberries.

- Avoid pureed fruit (raises insulin levels more than whole fruit).
- Avoid fruit juice. Small amounts of fruit juice are okay as a recipe ingredient. An occasional small “splash” of juice in water, seltzer, or iced tea is okay.

Limit Quantity:

- Allium family, thoroughly cooked only. The allium family includes onions, garlic, leeks, scallions, shallots, and chives. Thoroughly cook these vegetables until they are very soft and the “bite” of their sulfurous chemicals is gone.
- Potatoes,* peeled and cooked only. Do not eat potato skins. Peel potatoes, taking care to remove any eyes, sprouts, green spots, and damaged or rotting areas. Cook thoroughly. (This information applies to all types of potatoes except for sweet potatoes and yams.)
- Crucifers, cooked only. Limit to one serving per day. If you have chronic constipation, you may want to avoid crucifers altogether as an experiment. The crucifer family includes:
 - Arugula (rocket)
 - Bok choy
 - Broccoli, broccolini, broccoli rabe
 - Brussels sprouts
 - Cabbages of all kinds, including savoy cabbage, Chinese/napa cabbage
 - Cauliflower
 - Collard greens
 - Cress
 - Horseradish
 - Kale
 - Kohlrabi
 - Maca
 - Mizuna

- Mustard greens
- Radish
- Rutabaga
- Swiss chard
- Tatsoi
- Turnip
- Wasabi
- Watercress

*These vegetables have a higher glycemic index and may cause blood sugar spikes, especially if roasted or baked (boiling and stir-frying are somewhat better).

Vegetable Precautions

- Be careful with raw vegetables. If you have a sensitive digestive system, particularly if you are prone to constipation, be aware that raw vegetables are harder to digest; therefore eating too many raw vegetables may bother you.
- Do not eat bitter vegetables. Eat vegetables while they're still fresh (or keep frozen vegetables frozen until right before use). Don't let them sit in the refrigerator for more than a week or so because some vegetables can become bitter with time, indicating they are higher in defensive toxins, some of which could make you ill.
- Be careful with seeds and skins. If you have a sensitive system, peeling vegetables and removing any seeds may help you tolerate them better.
- Be careful with fiber, particularly if you are prone to bloating, constipation, or abdominal pain. While a common recommendation under these circumstances is to increase your fiber intake, I suggest you try the opposite.

Seasonings

- Salt
- Leafy and floral herbs of all kinds are permitted (basil, parsley, dill, thyme, cilantro, tarragon, etc.).
- Peppercorns of any color (black, white, pink, etc.) are permitted, but not spices derived from true peppers in the nightshade family such as paprika, cayenne, chili powder, red pepper flakes, etc.
- Vanilla extract (in alcohol, not glycerol)
- Lemon and lime juice; small amounts of rind are okay.
- Vinegars, tart only; avoid sweet balsamic vinegars.
- Infused olive oils such as (dairy-free) butter olive oil, lemon olive oil, etc., are okay so long as the infusion source is on the approved list (avoid garlic-infused oils, for example).
- Avoid spices derived from seeds, bark, and roots such as mustard, cinnamon, nutmeg, coriander, ginger, etc.

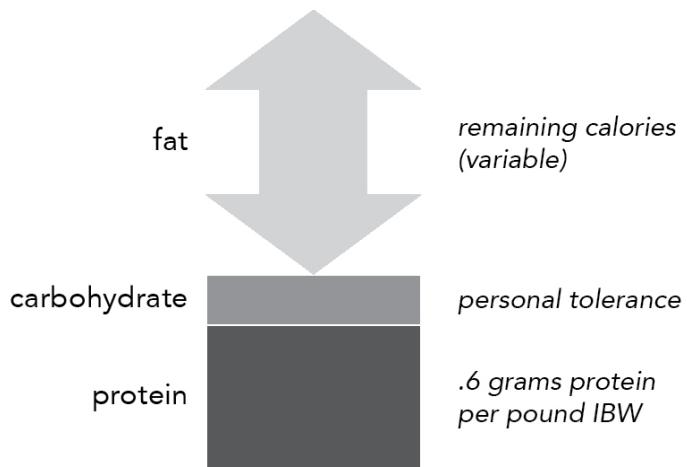
Beverages

- Water and unsweetened seltzer (flavored with natural fruit essences if you like). You can make your own flavored, infused waters, seltzers, and iced teas using citrus, small pieces of fruit, mint, basil, or cucumber and refrigerating overnight.
- Unsweetened coffee and tea (see [chapter 20](#) for notes about caffeine)

HOW MUCH SHOULD YOU EAT?

Now that you know *what* to eat, it's time to think about *how much* to eat. Food quantity is just as important as food quality, because overeating or undereating can make even the healthiest diet unhealthy. Fortunately, it's harder to overeat Quiet Paleo foods because they are naturally satisfying and nonaddictive, but if you have a history of food addiction or food restriction, you may have a tendency to overeat or undereat no matter what plan you follow. Protein, fat, and calorie requirements vary from person to person, and some people can safely handle more carbohydrate than others, so understanding how to tailor your macronutrients to your specific needs is the key to success with any diet.

You can use the simple calculations below to estimate your macronutrient targets, or if you prefer not to do this yourself, you can consult with a nutrition professional to personalize your plan. If you have a healthy relationship with food and you have always easily maintained a stable, healthy weight, you can probably trust your appetite and may not need to intentionally manage your protein, fat, and calorie intake when following Quiet Paleo, but it's still helpful to have a sense of what your healthy target ranges should be. You can download a free Quiet Diet Macronutrient Guide from my website that lists how many grams of protein, fat, and carbohydrate are in each food, but I've provided some ballpark estimates below to get you started.



MACRONUTRIENT GOALS

1. Prioritize Protein

To estimate your protein needs, you'll first need to estimate your *ideal body weight* (IBW) by using these simple formulas:

Women (assuming medium frame, average muscle mass): 100 pounds for the first five feet of height plus 5 pounds for every inch over five feet. For example, if you are five feet three inches tall, your estimated IBW would be 100 pounds + 15 pounds = 115 pounds. If you're under five feet tall, subtract 2 pounds per inch.

Men (assuming medium frame, average muscle mass): 106 pounds for the first five feet of height plus 6 pounds for every inch over five feet. For example, if you are five feet ten inches tall, your estimated IBW would be

$106 \text{ pounds} + 60 \text{ pounds} = 166 \text{ pounds}$.

Note that IBW is an imperfect concept based on gender, height, and frame; if your estimated IBW looks too high or low for you, simply use your own best estimate of what you believe your IBW to be.

Next, use your IBW to estimate your daily protein requirements. Protein recommendations vary widely among experts from between 0.4 and 0.75 grams of protein per pound IBW. I recommend using **0.6 grams of protein per pound IBW** as your starting point. For example, if your IBW is 125 pounds, multiply 125 by 0.6 to arrive at your estimated daily protein target, which would be 75 grams of protein per day.

TIP: One large egg contains 6 grams of protein. Most cuts of meat, seafood, and poultry contain between 5 and 8 grams of protein per ounce, depending on how lean or fatty they are (lean meats contain more protein per ounce).

2. Customize Carbohydrate

Monitoring your own blood sugar levels will help you figure out how much carbohydrate you can safely tolerate. Carbohydrate tolerance varies greatly from one person to the next, but most of us now have some degree of insulin resistance and can't safely process the amount of carbohydrate in a "normal" diet. (The U.S. Dietary Guidelines recommend that 45 to 65 percent of your calories come from carbohydrate, which on a 2,000 calorie per day diet translates to between 225 and 325 grams of carbohydrate per day.) This is why Quiet Paleo starts at about **90 grams of low glycemic carbohydrate per day**. Spread your carbohydrates out over the course of the day to minimize the risk of unhealthy glucose and insulin spikes. For example, if you eat three meals a day, simply include about 30 grams of carbohydrate from the Quiet Paleo kinder, gentler fruits and vegetables list in each meal. This will usually work out to about one serving of non-starchy vegetables plus up to one serving of a starchy vegetable or one piece of fruit per meal. Unlike ketogenic diets, you don't have to be precise about your carbohydrate content. Non-starchy vegetables such as asparagus, cucumbers, and lettuce are very low in carbohydrate, so you can enjoy a serving of these at every meal if you like.

TIP:

Strawberries = 10 grams/cup; raspberries and blackberries = 15

grams/cup; blueberries = 20 grams/cup

One apple, banana, orange, or pear = 25 grams each

One sweet potato or one cup of butternut squash = 25 grams each

One white potato = 35 grams each

Quiet Paleo begins at 90 grams of carbohydrate per day to prevent you from going into ketosis. However, if you have a significant degree of insulin resistance, 90 grams may be too high for you, so keep an eye on your glucose readings to be sure your after-meal glucose readings stay in a healthy range. Home glucose monitoring will be the key to discovering your personal “sweet spot.” If your glucose rises out of the healthy range, reduce your daily carbohydrate intake by 5 to 10 grams per meal and then reassess. Do not go below 60 grams per day while following Quiet Paleo because you might enter ketosis, which isn’t the goal of this phase of your discovery process.

3. Fat Is Flexible

The remainder of your calories should come from fat, and the amount you should eat will depend on how much energy (calories) you need. Human protein requirements fall within a relatively narrow range, and most of you will need to keep carbohydrate fairly low to achieve the results you’re looking for, so fat is where there is the most room for flexibility. I recommend you avoid counting calories or fat grams during the first six weeks of Quiet Paleo and let your appetite be your guide. In other words, begin by eating as much fat as you need to feel satisfied. It’s common to feel hungrier than usual in the beginning of your journey as your metabolism shifts gears, so it may feel like you’re overeating fat at first, but once your glucose and insulin levels quiet down, your appetite will usually quiet down, too. If you’re losing weight that you don’t want or need to lose, you’re losing weight too fast, or you’re hungry between meals, increasing your fat intake will often solve the problem. It’s hard to overeat fat on a Quiet Paleo diet because it is free of dairy and nuts (the types of fat people find most addictive), but if you’re gaining unwanted weight, you may need

to eventually cut back on your fat intake.

KEEP IT SIMPLE OR GET CREATIVE

The beauty of Quiet Paleo is that you don't need to intentionally limit protein, fat, or calories, because it's not meant to be a diet, it's meant to be a healthy way of life. All you need to do is stick to the approved food list, keep your fruit and starchy vegetable intake to one serving per meal, and monitor your blood sugar. If you enjoy cooking and want creative inspiration, you can find meal plans and recipes for Quiet Paleo in [chapter 21](#), but creating a Quiet Paleo meal can be as simple as choosing a source of animal protein, adding some non-starchy vegetables cooked in fat or drizzled with oil, and having a piece of fruit for dessert. Here's an example of how easy it can be to put meals together (the below sample menu provides 74 grams of protein):

Breakfast: 3-egg vegetable omelet (18 grams of protein) cooked in 1–2 tablespoons of duck fat, one apple

Lunch: Salad (made of kinder, gentler vegetables) with olive oil, lemon juice, avocado, and herbs topped with 5 ounces of grilled salmon (30 grams of protein); one cup of blueberries for dessert

Dinner: 2 roasted chicken legs (26 grams of protein), one cup of steamed zucchini, and a baked sweet potato topped with natural juices from the roasted chicken.

Most people will benefit to some extent from this relaxed plan, but you may eventually need to make adjustments to suit your metabolism. If your blood sugar and insulin levels are in good control but you're gaining unwanted weight, cut your fat intake down a bit. If your blood sugar runs too high after meals, cut your carbohydrate intake down a bit (without going below 60 grams of carbohydrate per day). If your blood sugar still runs high at 60 grams of carbohydrate per day, or if your mental health hasn't improved enough, I would encourage you to consider a ketogenic diet.

CHAPTER 18

Quiet Keto

Ketosis fundamentally changes the way your brain operates, unlocking your potential to experience improved mood, mental clarity, emotional resilience, and peace of mind. In this chapter, you will learn how to determine whether a ketogenic diet is appropriate for you and how to make your transition to ketosis as comfortable as possible. Remember, before trying any ketogenic diet, follow Quiet Paleo for at least two weeks to allow your glucose and insulin levels to come down more gradually.

LOOK BEFORE YOU LEAP

Ketogenic diets are powerful metabolic interventions that can lower blood sugar, blood pressure, and insulin levels very quickly. These are healthy changes, but if they happen too fast, it can be uncomfortable or even potentially dangerous—particularly if you are taking medications that lower blood sugar or blood pressure. Combining medicines such as these with a ketogenic diet could result in dangerously low blood glucose and/or blood pressure, so it's critically important that you notify the professional who prescribes those medicines for you *well before* you begin this diet to make sure they are willing and available to work with you to order laboratory tests, monitor your symptoms, and make any necessary medication adjustments.

I recommend both medical and psychiatric supervision while transitioning to a ketogenic diet, especially if you are taking prescription medications of any kind, have any pre-existing medical conditions, or have ever had any serious mental health symptoms such as suicidal thinking, self-injury behaviors, manic episodes, psychosis, dissociation, aggressive thoughts or behaviors, or bouts of confusion. Even if you haven't had these

symptoms lately, there is a small chance that they could temporarily resurface during the first few weeks of the ketogenic diet as your system finds its new equilibrium. Your safety is of the utmost importance.

This book is not intended to be your only guide to ketogenic diets for mental health; it is meant to be a source of information to support you and your health care providers and increase your chances of success. If you don't have access to appropriate health professionals, see appendix B for suggestions.

You should not use this chapter to start a ketogenic diet if you are:

- Under the age of 18
- Underweight (BMI <20); BMI stands for “body mass index”; see appendix B for link to calculate your BMI
- Pregnant or breastfeeding
- Taking an SGLT2 inhibitor such as canagliflozin (Invokana), dapagliflozin (Farxiga), or empagliflozin (Jardiance)

You should not use this chapter to start a ketogenic diet if you have any of the following conditions:

- Anorexia nervosa
- Mental health crisis situations such as new or worsening psychosis, suicidal thoughts, mania, agitation, aggressive or violent thoughts, confusion, recent traumatic experience
- Active substance abuse
- Acute injury or medical illness (influenza, burns, appendicitis, head injury, COVID-19, pneumonia, etc.)
- Kidney failure
- Acute pancreatitis
- Porphyria
- Rare genetic disorders of fat/ketone metabolism (usually diagnosed in infancy) that interfere with the body's ability to use fat and/or ketones for energy. These include glycogen storage disease type I, primary

carnitine deficiency, carnitine palmitoyltransferase deficiency types I and II, carnitine translocase deficiency, pyruvate carboxylase deficiency, succinyl-CoA acetoacetate transferase deficiency 2, acyl-CoA dehydrogenase deficiencies, beta-ketothiolase (T2) deficiency, methylmalonyl CoA epimerase deficiency, and 3-hydroxyacyl-CoA deficiency

Consult with a specialist first if any of the following situations apply to you:

- Diabetes (type 1 or type 2)
- Heart disease
- History of bariatric surgery
- Pancreatic disease
- Kidney disease
- Liver disease
- Gallbladder disease/no gallbladder
- Prone to kidney stones
- Gout
- Taking medications for diabetes, high blood pressure, or heart disease
- Cancer (the book *Keto for Cancer* by Miriam Kalamian¹ is a comprehensive and compassionate guide)
- Epilepsy (the Charlie Foundation website is a treasure trove of information)
- You are an elite athlete (see the work of Dr. Jeff Volek, Dr. Stephen Phinney, Professor Tim Noakes, and Dr. Caryn Zinn for guidance)

Connect with your Clinician(s)

Contact your medical and psychiatric care providers to let them know you'd like to try a ketogenic diet and ask if they'd be willing to help you. In addition to your medical and psychiatric practitioners, you also have the option of involving a dietitian or nutritionist who is knowledgeable about ketogenic diets to help personalize your eating plan, and/or a keto coach or

counselor for guidance and support (see appendix B for help locating keto-savvy clinicians).

If your medical and psychiatric clinicians aren't supportive, ask them to share their concerns with you. If their concerns have to do with the particulars of your medical or psychological situation, hear them out. If you disagree with their assessment, gather feedback from others you trust or request a second opinion.

If your clinicians are generally uncomfortable with the idea of ketogenic diets or view ketogenic diets as unhealthy or unsafe for *everyone*, ask if they are open to learning more—there are resources for health professionals listed in appendix B. If they're not interested or don't have time, you may need to seek out new health care providers or consultants, especially if you take prescription medications or have any serious pre-existing health conditions such as diabetes, high blood pressure, or heart disease.

ESTIMATE YOUR KETO MACROS

The process of determining your macronutrient requirements for a ketogenic diet is essentially the same one outlined in the previous chapter, with the main difference being that you'll need to lower your carbohydrate intake enough to allow your metabolism to shift into ketosis. Your age, metabolic health, activity level, pre-existing medical conditions, body composition, stress level, medications, and health goals all help determine your personal carbohydrate limit, protein requirements, and ideal fat intake. Use the principles below to estimate your macronutrient starting points and adjust from there as needed. If you prefer to use an online tool to calculate your estimated macros, please see appendix B for suggestions.

Prioritize Protein: Your protein requirements won't be any higher on a ketogenic diet than on a paleo diet, so use your estimated protein target (see [here](#)) as your starting point and adjust from there if needed.

Customize Carbohydrate: Like many ketogenic diets, Quiet Keto starts at approximately 20 grams of carbohydrate per day, because that is low enough to allow most people to shift into ketosis. However, since carbohydrate tolerance varies, some people may need to lower their carbohydrate intake even further to produce ketones, while others can get away with well over 20 grams per day and remain in ketosis, particularly

athletes and people whose jobs are physically demanding, because working muscles draw glucose out of the blood and burn it for energy. Since carbohydrates on Quiet Keto come almost entirely from fruits and vegetables, your carbohydrate tolerance will determine the quantity of fruits and vegetables you can safely include. Most people will need to limit their non-starchy vegetable intake to 2 to 3 cups per day spread over the course of the day and keep fruit intake very low (a handful of berries, for example).

There are two popular ways to count carbohydrate grams:

- *Total* carbohydrate grams encompass all digestible and indigestible carbohydrates including sugars, starches, fiber, and sugar alcohols
- *Net* carbohydrate grams include only the fully digestible carbohydrates (total carbohydrate minus fiber and sugar alcohols).

The meal plans in this book track total carbohydrate grams minus fiber grams (but do not subtract sugar alcohols). If you find you have trouble achieving ketosis using this definition, try counting total carbohydrate grams instead.

Figure in Fat: The remainder of your calories should come from fat. In most cases, there's no need to intentionally count or limit fat calories because a well-formulated ketogenic diet tends to regulate appetite nicely, making it easier to trust your instincts about how much to eat. It's uncommon to gain unwanted weight if you're in ketosis, because your insulin levels aren't high enough to turn fat storage enzymes on.

TIP: One tablespoon of fat (of any kind) = 14 grams of fat (~120 calories)

MACRONUTRIENT CALCULATION EXAMPLE

Protein and carbohydrate each contain 4 calories per gram, and fat contains 9 calories per gram.

If your IBW is 125 pounds, then your estimated daily protein requirement will be 75 grams (300 calories). Quiet

Keto caps daily carbohydrate at 20 grams (80 calories). Therefore, protein + carbohydrate calories = 380 calories per day.

The rest of your calories should come from fat. If you require 1,800 calories per day, 1,800 calories minus 380 calories = 1,420 calories of fat. Since one tablespoon of fat contains about 120 calories, that works out to about 12 tablespoons of fat per day, or 4 tablespoons (1/4 cup) per meal. However, since all whole food sources of protein naturally contain some fat already, adding 2 to 3 tablespoons of fat to each meal is plenty, and may even be more than enough if you are eating very fatty cuts of meat or including fatty plant foods such as avocados.

QUIET KETO FOOD LIST

Since the Quiet Keto diet is simply a very-low-carbohydrate version of the Quiet Paleo diet, the only difference between the two food lists is that the Quiet Keto list excludes high-carbohydrate fruits and starchy vegetables.

Meat, Seafood, Poultry, and Eggs

- Stay within your daily protein allowance for best results. No need to trim fat or remove skin. Minimize aged, cured, smoked, and processed meats because they can be high in histamine (see [chapter 19](#)).

Non-Starchy Vegetables (count non-fiber carbohydrate grams and stay under your personal limit)

- Lettuces: all true lettuces are permitted, including iceberg, romaine, Bibb, Boston, green leaf, red leaf, oak leaf, Batavia, and butterhead. Avoid non-lettuce salad greens such as raw spinach leaves and raw cruciferous greens such as arugula, kale, raw cabbage, radicchio, mustard greens, and watercress.

- Endive
- Cucumbers
- Mushrooms
- Globe artichokes
- Sunchokes (aka Jerusalem artichoke)
- Spinach (cooked)
- Celery
- Asparagus (cooked)
- Fennel
- Jicama
- Water chestnuts

Fruits

Note: Fruits lowest in carbohydrate are listed below, but so long as you count their carbohydrate grams and stay under your personal daily carbohydrate limit, any fruit except goji berries, wolfberries, and star fruit are permitted.

- Avocado
- Olives
- Squashes: zucchini, yellow squash, summer squash, pumpkin, and spaghetti squash
- Strawberries
- Blackberries
- Raspberries
- Lemons
- Limes

Special Vegetable Considerations

- Allium family, cooked only. The allium family includes onions, garlic, leeks, scallions, shallots, and chives. Always thoroughly cook these vegetables until they are very soft and the “bite” of their sulfurous

chemicals is gone.

- Crucifers, cooked only. Limit to one serving per day. If you have chronic constipation, you may want to avoid crucifers altogether as an experiment. The crucifer family includes:
 - Arugula (rocket)
 - Bok choy
 - Broccoli, broccolini, broccoli rabe
 - Brussels sprouts
 - Cabbages of all kinds, including savoy cabbage, Chinese/napa cabbage
 - Cauliflower
 - Collard greens
 - Cress
 - Horseradish
 - Kale
 - Kohlrabi
 - Maca
 - Mizuna
 - Mustard greens
 - Radish
 - Rutabaga
 - Swiss chard
 - Tatsoi
 - Turnip
 - Wasabi
 - Watercress

Seasonings

- Salt, including gourmet flavored salts
- Leafy and floral herbs of all kinds (basil, parsley, dill, thyme, cilantro, tarragon, etc.)
- Peppercorns of any color (black, white, pink, etc.) are permitted, but

not spices derived from true peppers in the nightshade family such as paprika, cayenne, chili powder, red pepper flakes, etc.

- Vanilla extract and other natural extracts are okay so long as they are sourced from foods on the approved list
- Lemon and lime juice (and small amounts of rind are okay)
- Vinegars, tart only; avoid sweet balsamic vinegars
- Infused olive oils such as (dairy-free) butter olive oil, lemon olive oil, etc. are okay so long as the infusion source is on the approved list (avoid garlic-infused oils, for example)
- No pungent spices derived from seeds, bark, or roots (mustard, cinnamon, nutmeg, cumin, coriander seed, ginger, etc.)

Beverages

- Water and unsweetened seltzer (flavored with natural fruit essences if you like). You can make your own flavored, infused waters, seltzers, and iced teas using citrus, small pieces of fruit, mint, basil, or cucumber and refrigerating overnight.
- Unsweetened coffee and tea (see [chapter 20](#) for notes about caffeine)

Following Quiet Keto for at least six weeks is the most efficient way to maximize your results, but if the idea of giving up dairy or nuts for that long is a non-starter and you would prefer a diet with a broader food list, feel free to use a different ketogenic diet, just make sure it contains adequate protein and that it's based on healthy whole foods. I recommend reading "The Ten Defining Characteristics of a Well-Formulated Ketogenic Diet" on the Virta Health website,² written by Dr. Stephen Phinney and Dr. Jeff Volek, and referring to the ketogenic diet resources in appendix B.

KETONE MONITORING

Measuring ketones isn't mandatory, but I strongly recommend it, especially early on, because it will help you get a feel for how your eating plan affects your metabolism, and how your metabolism influences your mental health.

You may recall from [chapter 5](#) that when you are burning fat, your liver

breaks down that fat into three different types of ketones: acetoacetate, acetone, and beta-hydroxybutyrate (BHB). Acetoacetate is unstable in the bloodstream, so some of it escapes into your urine and some of it spontaneously turns into acetone (which you exhale).

Blood Ketone Monitoring: The most accurate way to measure ketones is with a blood meter, which measures BHB. (Choose a meter that measures both ketones and glucose, such as the Keto-Mojo GK+.) Testing requires that you prick your finger (or toe); however, measuring once a day is usually sufficient, and once you learn how to keep yourself in ketosis, you can measure less frequently.

Urine Ketone Monitoring: Urine testing (which measures acetoacetate) is easy, inexpensive, and painless. The darker the color on the test strip, the higher the acetoacetate level in your urine. This method is imprecise, because the amount of acetoacetate in your urine will vary depending on many things, including how hydrated you are, how long it's been since you last emptied your bladder, and how efficiently your body is using acetoacetate. Urine testing essentially measures how much acetoacetate your body has lost since you last emptied your bladder, so it can't tell you whether you are in ketosis in real time. However, urine strips will give you a "yes-no" answer about whether you've been in ketosis over the past few hours, and that is very useful information.

Breath Ketone Monitoring: Breath testing (which measures exhaled acetone) is painless, and while the meter itself is expensive, it doesn't require test strips so it can save you money over time, and you can test as often as you want.

All of these testing methods give you valuable information, but I recommend blood testing if you can afford it, because it is most accurate and provides real-time data. Some people like to combine once-daily blood testing with more frequent urine or breath testing because they are curious to know what is happening to their ketone levels throughout the day. If you are using a blood meter and testing only once a day, I recommend testing before your first meal of the day, while you're still fasting. (TIP: If you can extend your overnight fast by delaying breakfast for at least two hours, that will give your brain a little more healing time each day.) Testing before your first meal gives you the cleanest baseline for comparing ketone levels from one day to the next, because your food choices, stress levels, and

exercise patterns can cause your ketone levels to vary quite a bit from one day to the next (which is normal). Also, ketones tend to rise as the day goes on, so if you only test later in the day, you won't know if you've been in ketosis all day long or only the second part of the day. (Many people feel better if they are in ketosis all day.)

Ketone Target Range

See [chapter 9](#) for a refresher on this topic, but I recommend you begin by aiming for blood ketone levels of between 1.0 and 3.0 mM and try to stay within that range most of the time every day for at least six consecutive weeks. Breath meters measure acetone in PPM (parts per million); therapeutic ketosis corresponds to levels ranging between 10 and 40 PPM. If you're using urine strips, aim for moderate to large ketones (medium to dark color). Some people find they need to keep their ketones within a particular range to feel their best while others are less sensitive to ketone levels. As you gain more experience with the diet, you'll get a feel for what your personal therapeutic range is. If you are achieving your goals despite ketone levels running on the low side, there is no need to try to raise them —treat your symptoms, not your ketones!

THE LONGEST MILE: KETO-ADAPTATION

Getting into ketosis can take as little as three days, depending on your macros and the particulars of your metabolic situation such as age, physical fitness, health conditions, and medications—but just because you see a nice ketone reading on your meter doesn't mean your cells are efficient at burning them for energy yet. As your metabolism switches from burning mostly glucose to burning mostly fat, there can be a period of discomfort as your system adjusts and finds its new equilibrium. Some people are metabolically flexible and shift comfortably into ketosis (if you're under forty, physically fit, or your metabolism is relatively healthy, you're more likely to fall into the flexible category), whereas others may experience “keto flu” symptoms such as difficulty concentrating, irritability, headaches, carbohydrate cravings, lightheadedness, trouble sleeping, leg cramps, palpitations, low energy, poor exercise tolerance, nausea, diarrhea, or

constipation. These can last from a few days to about two weeks at most, but if you follow the recommendations in this chapter, transition symptoms will likely be very mild or may not occur at all.

As unfair as it is, some people with mental health conditions face additional challenges as they transition to ketosis. These may include the temporary worsening of anxiety or depression, the appearance of mood swings, or even hypomania (mild manic symptoms such as sleeplessness, increased energy, anxiety, and hyperactivity). In my experience these don't happen to most people, and when they do occur, they almost always resolve by week three. You can minimize and often avoid both keto flu and psychological discomfort by:

- Transitioning gradually to a ketogenic diet by following Quiet Paleo for at least two weeks first
- Actively replenishing electrolytes throughout the day (see below)
- Staying well-hydrated
- Keeping stress low
- Prioritizing sleep
- Going easy on exercise

If these strategies don't help enough, you might also consider briefly using a ketone supplement or MCT oil to support slightly higher ketone levels during the transition (see below).

If you experience any psychological symptoms during the transition that are too great to bear, interrupt the diet by having a whole-food, carbohydrate-rich snack such as a banana or half of a sweet potato and you should feel better within an hour or so. Take care of yourself, return to Quiet Paleo, and regroup. If transitioning to a ketogenic diet feels psychologically rough, that is not your fault; it usually just means that you and your health care provider(s) need to adjust your plan to make it more comfortable for you. This may involve the temporary use of supplements or medications, changes to the diet to ease you more gradually into ketosis, or troubleshooting your electrolyte plan.

Mind Your Minerals

High insulin levels tell your body to retain water and salt, which causes water weight gain and can lead to swelling in the lower legs, along with a general sense of heaviness and bloat. As insulin levels fall on a ketogenic diet, that excess sodium and water is flushed from the kidneys. This is a healthy change, but if your insulin levels fall too fast, your sodium levels may also fall too fast. If this happens, your adrenal glands (which sit on top of your kidneys) will perceive it as an emergency and tell your kidneys to hold on to more sodium so you won't lose too much too fast. However, the only way your kidneys can do that is by letting go of some potassium instead. This is why it is so important to take in extra sodium and potassium when you are adapting to a ketogenic diet.

Drink plenty of water and supplement electrolytes throughout the day. Do not overlook this piece of your plan as you could become dehydrated or develop mineral imbalances. Symptoms of mild mineral imbalances include lightheadedness, headache, difficulty concentrating, constipation, and leg cramps. Symptoms of more severe mineral deficiencies include palpitations and low blood pressure, which could cause loss of consciousness. Salt your food generously, and sip something salty throughout the day like bone broth, a homemade electrolyte drink (see below), or pre-mixed electrolyte supplements (a clean brand is LMNT raw unflavored, which contains no sweeteners). If you do experience symptoms of possible mineral imbalances, take some additional electrolytes and you will usually feel better within a few minutes. If electrolytes don't help, there is a chance your symptoms could be due to hypoglycemia, so test your blood glucose. If it is below 55 mg/dl, take one tablespoon of fruit juice immediately and retest 15 minutes later. If it is between 55 and 70 mg/dl, only treat with juice if you have symptoms such as nausea, shaking, sweating, dizziness, racing heart, confusion, extreme sleepiness, or poor coordination.

Estimated electrolyte requirements during keto-adaptation:

- 5 grams (5,000 mg) of sodium per day (roughly 2-1/2 teaspoons of sea salt)
- 4 grams (4,000 mg) of potassium per day (easiest to obtain from food)

and from salt substitutes made with potassium chloride)

- Magnesium, approximately 400 mg per day (try magnesium glycinate 200 mg twice a day or Slow-Mag Mg Muscle + Heart formulation, 2 to 3 tablets per day)

Once your biology finds its new equilibrium, you may not need to continue electrolyte replacement.

HOMEMADE ELECTROLYTE SOLUTION

Dissolve 2 teaspoons of Morton Lite salt (contains sodium and potassium) and 1/4 teaspoon of baking soda in 1 quart of water and sip throughout the day. A squeeze of fresh lemon juice and some crushed ice will make it more refreshing. You can continue the sodium and potassium for as long as you like but do not use the baking soda for longer than six weeks. (The pinch of baking soda helps prevent your blood from becoming slightly too acidic during the transition, which can happen in some cases.) Avoid using store-bought sports beverages to replenish your electrolytes because all of them contain sweeteners which could lower your ketone levels.

Note: If you have heart disease, high blood pressure, or kidney disease, consult with your health care team about electrolyte replacement.

What to Expect

Most keto flu symptoms resolve within the first week or two, but some symptoms can linger, especially low energy. Be patient; adaptation symptoms usually resolve by week six.

In terms of seeing changes and improvement to your mental health, the first three days are the hardest. Some people experience substantial benefits by day four. Most notice significant improvement by week three. If you don't feel better after six weeks, you can troubleshoot, try a different

approach, or consult a specialist. If you feel only partially better after six weeks, and you don't want to try Quiet Carnivore, I recommend continuing Quiet Keto for an additional six weeks to give your system more time to respond to the diet.

MEDICATION MANAGEMENT

Starting a ketogenic diet while taking psychiatric medication can feel more challenging than it otherwise would be, because:

- Some medications can raise glucose levels, raise insulin levels, and/or cause insulin resistance, making it harder to achieve ketosis.
- Shifting into ketosis can affect the blood levels of some medications.
- Combining certain medications with the ketogenic diet increases the risk for specific medical problems such as kidney stones.
- Once the diet takes effect, many medications can begin to feel too strong and cause potentially serious side effects as early as week three.

Quite a few non-psychiatric medications can also raise glucose levels or cause insulin resistance and make it more difficult to achieve ketosis, including prednisone, cholesterol-lowering statin drugs, and most liquid medications (because most contain sugar).

As yet, there are no published guides to managing psychiatric medications on a ketogenic diet. This is partly because the field is still so new that we don't yet have enough collective experience to produce a care consensus guideline, and partly because managing psychiatric medications is complicated under all circumstances, not just in the context of ketogenic diets. Complete coverage of medication management is beyond the scope of this book, so please consult appendix B for additional resources.

Some may look at all these special considerations and conclude that the ketogenic diet is too extreme or dangerous to be good for people with mental health issues, but most challenges that arise have to do with medications and pre-existing medical conditions, not the diet itself. Remember, medications are designed to interfere with normal biology. Your normal biology is evolutionarily designed to shift back and forth between a

glucose-based metabolic state and a fat-based metabolic state; it's the medications that can get in the way of that happening as it should. Depending on the medications you take, it could take longer to experience good appetite control or see satisfying ketone levels show up on your meter—but it is well worth the time and effort to make it to the other side. Once you are in ketosis and you've given your chemistry a few weeks to settle down, nine times out of ten, you'll be glad you stuck with it.

I hope you can appreciate the complexity of this issue and why it's so important not to go on this journey alone, especially if you take prescription medications. Even if you enter ketosis easily, sail through keto-adaptation, feel better on a ketogenic diet, and your medications haven't caused you any problems along the way, you will still need medical supervision if you want to try reducing the amount of medication you take. **Please, do not change your medication plan without professional guidance and never stop any psychiatric medication abruptly.** The vast majority of psychiatric medications must be tapered down slowly and carefully to minimize the risk of withdrawal symptoms. These can range from uncomfortable symptoms such as dizziness and electric shock sensations to life-threatening symptoms such as agitated depression or seizures, depending on the medication. There's no one-size-fits-all approach to tapering, so please consult with your prescribing clinician as needed.

The ketogenic diet doesn't help everyone reduce or eliminate their psychiatric medication; you and your psychiatrist or nurse practitioner will need to discover what's possible for you. However, even if Quiet Keto doesn't help you get *off* psychiatric medication, it could help you feel better *on* psychiatric medication by counteracting common side effects such as weight gain, sleepiness, and high blood sugar that reduce quality and length of life. If you have found certain psychiatric medications beneficial but don't like how they make you feel, the ketogenic diet could be a very healthy addition to your medication plan.

EXOGENOUS KETONES, MCT OIL, AND COCONUT OIL

If you're having a difficult time getting through keto-adaptation, the temporary use of supplements that support higher ketone levels may help. One option is to consume oils that your liver converts rapidly into BHB.

The other option is to take a ketone supplement that contains actual BHB.

MCT oil is purified, 100 percent *medium-chain triglycerides*. MCT products are industrially refined oils with no taste or odor. Most of the fats found in food are long-chain triglycerides, which take more time to break down into ketones. Because MCTs are shorter in length, the liver can turn them into ketones much faster, supporting slightly higher blood ketone levels for several hours. Look for MCT oils containing high amounts of caprylic acid (also referred to as “C8”) because caprylic acid raises ketones more effectively than other MCTs. The maximum recommended dosage is about two tablespoons three times per day, but MCTs can cause digestive upset, so start with one teaspoon at a time and try slowly working your way up. MCT powders, which blend easily into beverages without separating, are also available, but these are spray-dried with fillers, so read the label before buying, because some contain dairy, soy, or even refined carbohydrates such as maltodextrin and glucose syrup.

Coconut oil: About half of the fats in coconut oil are MCTs, but only a small percentage of these are short enough to turn rapidly into ketones, so coconut oil is a very weak ketogenic supplement.³ I don’t include coconut products in my Quiet Diets because coconuts are technically seeds, and therefore can be problematic for some people. However, if coconut oil doesn’t bother you, you could try adding some (unrefined) coconut oil to your ketogenic diet. The main benefits of coconut oil are that it is rich in saturated fat (which is very satisfying), has a pleasant taste, and is stable at cooking temperatures of up to 350 degrees Fahrenheit.

Ketone salts are BHB molecules bound to salts like sodium or calcium. They are pricey, and each dose lasts less than two hours in the bloodstream, making them too expensive for most people to take regularly. They don’t taste good, so most brands are sweetened and many also contain caffeine. The market is flooded with choices, some of which are of poor quality, so read labels carefully to make sure they contain a decent amount of BHB (better brands typically contain about 12 grams of BHB per dose). Measure your ketone level before you take them and forty-five minutes later to make sure they’re worth your money. All brands are high in sodium, so you do not need to supplement sodium when ketone salts are in your system.

Ketone esters are pure liquid BHB in a bottle. These chemicals are very expensive (because they are costly to isolate), and they taste perfectly

awful. They do raise ketones substantially—sometimes by as much as 3.0 mM—but, like ketone salts, their time in the bloodstream is short. They also spike ketones so effectively that your body may stop making its own ketones for the rest of the day. Despite these limitations, they can be useful for those who can't follow a ketogenic diet but who need the benefits of ketosis, such as people with cognitive impairment who can't manage or won't follow a ketogenic diet, and people who don't have much control over their food choices, such as people living in group homes, residential care facilities, or other institutional settings.

I don't generally recommend long-term use of ketone or MCT oil supplements to those following a ketogenic diet, because they're expensive, industrially refined products that don't last long in your system, and they're usually unnecessary. If you're following a well-formulated ketogenic diet, your body will naturally produce all the ketones you need, around the clock, for free. And no supplement can prevent high glucose and insulin levels; only diet can do that.

TROUBLESHOOTING KETONE LEVELS

Difficulty achieving or maintaining sufficient ketone levels is the most common reason for poor results. People often consult with me and tell me that the ketogenic diet didn't work for them, or that it made their mental health condition worse. In almost every case, it turned out that they either weren't in ketosis consistently enough, or that they had stopped the diet after just a few days because they didn't like how they felt on it. I'll tell you what I tell them: *If you haven't tried being in ketosis consistently for at least six weeks, you haven't tried it yet*, so you don't yet know what might be possible for you.

Ketones too low? If your ketones tend to hover well below 1.0 mM, look for common culprits such as hidden carbohydrates, insulin-spiking ingredients like sweeteners and protein powders (both of which are discouraged on Quiet Keto), overeating, or medications that raise glucose and/or insulin levels. Remember that it is the quantity of protein plus the quantity of carbohydrate in your diet that largely determines how high your insulin levels rise after eating, and the higher your insulin, the lower your ketones. If your ketones are too low, try lowering carbohydrate first by

limiting your carbohydrate to 20 *total* grams per day. Reduce protein only if other strategies haven't helped, and do not go below 0.40 grams of protein per pound of ideal body weight. Stress, sleep deprivation, injury, illness, and certain medications can also lower ketone levels.

Ketones too high? If your ketones run above 3.0 mM most of the time and it feels uncomfortable or you're losing weight quickly, you probably need more food. Common symptoms include insomnia, cravings, agitation, and feeling scattered. Try adding more protein to your plan first before you consider adding more carbohydrate.

Ketones all over the place? It's perfectly normal for ketones to fluctuate throughout the day and night, but the goal is to keep them between 1.0 mM and 3 mM most of the time. If you're repeatedly going in and out of ketosis, it will be hard for your metabolism to shift gears and adapt to burning fat for energy. This state of metabolic purgatory is what I call "no-man's land." This situation can be frustrating, and even physically and psychologically uncomfortable, because it can trap you in the keto-adaptation phase indefinitely.

Track your trends using a calendar and give yourself a star for every day that your ketones reach at least 1.0 mM. Aim to string together a full six weeks of daily stars. If you're going in and out of ketosis, start keeping a food and exercise journal to see if you can figure out what the problem is. If your ketones run low a few times during that six-week stretch, it's probably fine, but the more consistent you can be, the sooner your metabolism will make the shift to fat-burning mode, which is when people tend to notice major benefits. Once you've shifted solidly into that state, cravings for carbohydrates tend to quiet down or even disappear.

WHAT IF KETOSIS GETS INTERRUPTED?

If you intentionally or accidentally fall out of ketosis, your mental health symptoms could return rather quickly—sometimes within as little as twenty-four hours—just as if you had abruptly stopped a psychiatric medication. Work with your mental health care professionals *before starting the diet* to create a personalized plan B for such situations—especially if you're using the ketogenic diet to treat severe psychiatric symptoms. In these cases, you may want to have a fast-acting medication on hand for

temporary emergency use, or a crisis support plan in place to keep yourself safe until you are back in ketosis.

Being fat-adapted is a precious (and sometimes hard-earned) metabolic state. The longer you are out of ketosis, the more likely it is that you will have to go through keto-adaptation all over again, so it is in your best interest to find your way back to ketosis as soon as possible. Should “ketosis interruptus” occur for any reason, the simple strategies below are designed to reduce your glucose and insulin levels as efficiently as possible to jump-start the fat-burning process. If you have been out of ketosis for more than a week, or if these strategies cause significant emotional or physical discomfort, return to Quiet Paleo for at least a week before trying any of these again.

Fasting Jump-Start +/- Ketone Support: Fast until your ketones rise to at least 1.0 mM. Be sure to consume broth and/or electrolyte supplements, and drink plenty of water and/or unsweetened seltzer. You can supplement with exogenous ketones or add a little MCT oil to your broth if it makes fasting more comfortable or tolerable for you.

High-Fat Whole-Foods Jump-Start +/- Intermittent Fasting: Eat only from the foods on the list below until you’re satisfied:

- Eggs cooked in fat (no dairy please)
- Pork belly, pork butt, baby back ribs (or bacon if you tolerate it well)
- Lamb belly, lamb ribs
- Oily fish: Sardines, mackerel, fatty salmon, eel, herring, sable, etc.
- Avocado (maximum 1 per day)
- Broth, water

These foods are naturally ketogenic and satisfying because they all have a high fat-to-protein ratio and little to no carbohydrate, so you don’t have to count or weigh anything. If your ketones haven’t risen to at least 1.0 mM by day 5, stick to the same food list, but narrow your eating window by eating only once or twice a day and fasting overnight for 14 to 16 hours.

Exercise Boost: Exercise reduces liver glycogen levels, shortening the time it takes to start burning fat. If you’ve had a higher-carbohydrate day and want to get back on track the very next day, do some high-intensity

exercise that night and/or the next morning and you'll turn the corner sooner.

BEYOND SIX WEEKS

The plans in this book are intended as short-term discovery strategies of six to twelve weeks to start you on the path to better metabolic health and better mental health. You and your clinician(s) can then use what you've learned to design your own personalized dietary road map going forward.

If Quiet Keto works well to control your mental health symptoms (which would be wonderful!), you can continue the plan as is; but if you long for a more diverse diet, you can try carefully expanding your food list as described in [chapter 16](#) to include cured meats, nuts, and/or a greater variety of (low-carbohydrate) vegetables and seasonings.

The majority of my patients who improve on a ketogenic diet do report a return of their psychiatric symptoms if they stop the diet. However, a small number of people seem to experience enough healing after being on a ketogenic diet for a year or two that they can increase the amount of carbohydrate in their diet without their original psychiatric symptoms recurring. This scenario may be more likely if you are young, metabolically healthy, physically fit, or have milder mental health concerns. The safest way to test the waters is to slowly increase your daily carbohydrate allowance (from fruits and vegetables) while monitoring your glucose levels and your mental health symptoms to see how much carbohydrate is safe for you.

If you'd like to continue on a ketogenic diet long-term, rest assured. All available evidence supports the idea that a well-formulated whole-foods ketogenic diet that provides all essential nutrients and keeps your ketones in a moderate range should be safe long-term, and there is no biological reason to think otherwise. Dramatic headlines warning that ketogenic diets may improve your health now but will endanger your health later all come from nutrition epidemiology and are therefore baseless. The other common criticism is that there are no long-term studies of ketogenic diets, so it's risky to follow them for extended periods of time. Proving beyond a reasonable doubt that *any* dietary pattern is guaranteed safe for everyone to follow for the rest of their lives is impossible, so no dietary pattern can

promise you this. Just as you would with any diet, continue with routine medical checkups and let your progress be your guide.

However, given everything we know about human metabolism, nutrition, and chronic disease, controlling blood sugar and insulin levels may be the single most powerful thing you can do to improve and protect your mental and physical health in the long run, and the ketogenic diet is a very effective way to accomplish this.

CHAPTER 19

Quiet Carnivore

If Quiet Paleo or Quiet Keto didn't bring you the benefits you'd hoped for, you may benefit from a carnivore diet, at least as a short-term experiment. As mentioned in [chapter 16](#), carnivore diets include a range of eating patterns from plant-free, all-meat diets to mostly-meat diets that include dairy, eggs, spices, alcohol, and sometimes even honey (because it is produced by bees and is therefore an animal product). At first glance, carnivore diets may seem extreme, irrational, and dangerous. However, when we look objectively at the biological differences between plant and animal foods, it becomes easier to appreciate the potential health benefits of these unorthodox ways of eating.

WHY CARNIVORE?

Plant-free diets efficiently eliminate multiple culprits all at once—grains, legumes, sugar, starch, fiber, all plant toxins, and nearly all antinutrients (eggs and dairy contain a few antinutrients, as mentioned in [chapter 11](#)). Plants are hard on the body, so excluding all plant foods, at least for a while, may offer unique therapeutic advantages—particularly for those of us with compromised defenses who have lost the ability to safely and comfortably tolerate a wide variety of plant foods. I have found carnivore diets to be indispensable discovery tools in my clinical practice not only for identifying food sensitivities, but for resolving chronic constipation and IBS symptoms, quieting food addiction and binge eating, and breaking weight loss plateaus. The simplicity of carnivore diets is also wonderful for people who want to get into ketosis but feel too depressed or overwhelmed to memorize food lists, follow rules, or count carbohydrates.

The theory behind the carnivore approach is that a diet consisting

exclusively of animal foods is the healthiest, safest, and most nutritious diet we can eat. Those who advocate for carnivore diets view plant foods as unnecessary, nutritionally inferior, and potentially harmful to human health. It's not possible to say with any scientific certainty whether this dietary pattern (or any dietary pattern, for that matter) is ideal for everyone, but my clinical and personal experience tells me that a well-formulated carnivore diet can be uniquely healing for some of us.

THE HISTORY AND SCIENCE OF CARNIVORE DIETS

Historical accounts exist of people eating mostly-meat or all-meat diets for long stretches of time without incident, including the Inuit hunters living in the Canadian Arctic, where plant foods are scarce. As Gary Taubes writes in his superb book *Good Calories, Bad Calories*:

The Inuit paid little attention to the plants in their environment “because they added nothing to their food supply,” noted the Canadian anthropologist Diamond Jenness, who spent the years 1914–16 living in the Coronation Gulf region of Canada’s Arctic coast. Jenness described their typical diet during one three-month stretch as “no fruit, no vegetables; morning and night nothing but seal meat washed down with ice-cold water or hot broth.” (The ability to thrive on such a vegetable- and fruit-free diet was also noted by the lawyer and abolitionist Richard Henry Dana, Jr., in his 1840 memoirs of life on a sailing ship, *Two Years Before the Mast*. For sixteen months, Dana wrote, “we lived upon almost nothing but fresh beef; fried beefsteaks, three times a day... [in] perfect health, and without ailings and failings.”)¹

Modern carnivore diets are such a new concept that no rigorous clinical trials yet exist exploring their effects on human health, but curious scientists are beginning to dip their toes into the water. In 2021, a social media survey conducted by Dr. Belinda Lennerz and colleagues at Boston Children’s Hospital of more than 2,000 adults who reported having followed a

carnivore diet for at least six months found that 95 percent were satisfied both with the diet itself as well as with its health benefits. With respect to mental health in particular, the majority reported improvements in food cravings (91 percent), energy (89 percent), mental clarity (85 percent), focus (83 percent), and sleep (69 percent). Of the 479 people with psychiatric concerns, 48 percent reported complete resolution of symptoms, 48 percent reported improvement in symptoms, and only 4 percent reported that the diet had had no effect. While these trends are encouraging, keep in mind that this information comes entirely from volunteer responses; no concrete data (such as blood tests, vital signs, or formal psychiatric evaluations) were collected. Also, the survey likely attracted people who were doing well on the diet and were therefore positively biased toward it, because only those who had been on the diet for at least six months were eligible to participate.

Internal medicine physician Dr. Csaba Tóth and neurobiologist Dr. Zsófia Clemens direct the International Center for Medical Nutritional Intervention in Hungary, where they prescribe all-meat diets to people with advanced, treatment-resistant medical conditions such as cancer and autoimmune diseases. Their protocol begins with a diet exclusively of meat and fat (from four-legged animals only) that can later be expanded as tolerated to a paleolithic ketogenic diet—a meat-based plan that allows select plant foods. Their view is that chronic diseases stem largely from increased intestinal permeability (leaky gut) which can be healed with their signature carnivore plan. They have published several interesting case reports including cases of type 1 diabetes (an autoimmune disease that eventually destroys the ability of the pancreas to produce insulin) in which insulin production capacity was preserved (preventing the need for insulin injections), and multiple cases of cancer in which tumor growth was halted for two years or more without the use of radiation or chemotherapy.²

DO PLANT-FREE DIETS CONTAIN ENOUGH NUTRIENTS?

Most nutrients are more abundant and more bioavailable in animal foods than plant foods, but there are important exceptions. In an article published in the journal *Current Opinion in Endocrinology, Diabetes, and Obesity*,

nutrition science writer and carnivore diet expert Amber O’Hearn addresses the question of whether a plant-free diet can meet our daily requirements for all essential nutrients.³ She begins by explaining that nutrient requirements are *context-dependent* (meaning they vary depending on what we eat), and since standard recommendations regarding vitamin and mineral intake were determined in the context of a typical (relatively high-carbohydrate) diet, the same rules do not apply. Nevertheless, she concludes that it is possible to meet standard nutrient requirements without eating plants, but that you may need to include specific animal foods to do so, most importantly liver. Liver is a rich source of vitamin A, which we associate with orange vegetables like carrots, and folate (vitamin B9), which we associate with leafy greens. Although the word “folate” comes from the Latin *folium*, which means leaf, ounce for ounce, liver is richer in folate than spinach (considered one of the best plant sources of folate)—just take care not to overcook liver, as folate is easily destroyed by heat.

When we think of vitamin C, we think of citrus fruits, not meat; if you recall from [chapter 3](#), it was citrus that rescued sailors from the ravages of scurvy. However, O’Hearn points out that it’s been known for centuries that fresh meat alone can cure scurvy, too, suggesting that the small amounts of vitamin C present in animal foods appear to be sufficient for our needs. Like folate, vitamin C is sensitive to heat so be sure not to overcook your food. You may want to include occasional raw choices such as sashimi, tuna tartare, or beef tartare. If you are worried you’re not getting enough vitamin C, add a squeeze of fresh lemon or lime juice to your water or seltzer if you tolerate it, or take a supplement.

As for calcium, it’s unclear whether you can meet your requirements unless you include dairy or certain kinds of seafood in your diet. If you don’t eat dairy (which I recommend you avoid), shrimp are a good source of calcium, as are small, soft-boned fish such as sardines, anchovies, and mackerel. If you are allergic to seafood or want to take a calcium supplement, be sure to choose one that contains vitamin D3 and vitamin K2 (and ideally magnesium as well, if you tolerate magnesium supplements), as these nutrients help your body absorb and utilize calcium properly.

The plans in this book are intended as short-term discovery strategies, so if you would like to follow a carnivore diet long-term, please work with a health care professional to periodically monitor your nutrient status and

your overall health to see if changes need to be made along the way. To be on the safe side, obtain a few nutrient blood tests prior to starting your carnivore diet to establish your baseline levels and then have them rechecked three months later. The nutrients most worth monitoring on a carnivore diet are vitamin A (best carnivore sources are liver and fatty fish), vitamin C (rare meats), folate (gently cooked liver), vitamin E (salmon, trout, goose), and vitamin D (sunshine and animal fats). Unfortunately, there are no good tests for vitamin K2 or calcium deficiency.

THE QUIET CARNIVORE DIET

Quiet Carnivore is a ketogenic diet, so please follow Quiet Paleo for at least two weeks before starting Quiet Carnivore, just as you would before embarking on any other ketogenic diet. Read [chapter 18](#) carefully before considering Quiet Carnivore to be sure a ketogenic diet is right for you. If you want to transition directly from Quiet Paleo to Quiet Carnivore without following Quiet Keto in between, that's perfectly fine, but you'll need to take all of the same preparatory steps and precautions outlined in [chapter 18](#) to ensure a smooth transition into ketosis.

The Quiet Carnivore diet grew out of my study of nutrition science and my experiences with patients who were struggling with food sensitivities and other health issues despite following other versions of a carnivore diet. The Quiet Carnivore food list consists primarily of fresh (or fresh-frozen) unprocessed meat, poultry, fish, shellfish, organ meats, bone broth, salt, and water/seltzer. Simplifying your diet to this degree allows you to start with a relatively clean slate which will help you determine if your mental or physical health symptoms may be rooted in food intolerances.

Quiet Carnivore excludes dairy and eggs, and minimizes processed animal foods that have been aged, cured, smoked, preserved, or fermented. Examples of products to limit include bacon, dry-aged beef, smoked salmon, hot dogs, jerky, ham, salami, and cold cuts.

Like all Quiet Diets, Quiet Carnivore eliminates dairy because it is such a common cause of inflammation, digestive problems, elevated insulin levels, increased appetite, and weight gain. Quiet Carnivore eliminates eggs because, despite the egg being a nearly perfect food, eggs are also a common cause of allergies and food sensitivities.⁴ Both eggs and dairy are

on the Food and Drug Administration's list of the "big nine" most common food allergies,⁵ and both can cause non-allergic sensitivity reactions in susceptible individuals as well.

FOOD SENSITIVITIES AND MENTAL HEALTH: A CASE EXAMPLE

Lisa is a nurse practitioner in her early sixties who had lived with frequent, debilitating panic attacks since the age of seventeen. In her twenties and thirties, she ate a vegetarian diet that included eggs and dairy products, but as she approached her forties her anxiety began to worsen, so she tried reintroducing meat, but it did not alleviate her symptoms. She tried talk therapy, cognitive-behavioral therapy, and EMDR (eye movement desensitization and reprocessing), as well as a variety of medications meant to treat depression and anxiety, including Celexa, Lexapro, Cymbalta, Prozac, Wellbutrin, and Zoloft. Talk therapy provided minimal support and the medications either didn't help or caused intolerable side effects. The one medication that brought any significant relief was Klonopin (clonazepam), which she'd been taking for fifteen years by the time we started working together.

Her health was also slowly deteriorating, and she had been diagnosed with chronic fatigue syndrome a few years after beginning the Klonopin. One day she was running late for an appointment and grabbed a hard-boiled egg to eat in the car on the way, having no time to eat or drink anything else, not even her usual cup of coffee. Within minutes, she had a full-blown panic attack. Through trial and error, she eventually figured out that eggs (specifically egg whites) were predictably causing every single one of her panic attacks. This, despite two rounds of allergy testing that revealed no food allergies. She told me:

When I think of the thousands of dollars I spent talking

to therapists about my childhood, only to realize that my panic attacks were caused by eggs, I really want to scream! I've now been free from daily Klonopin use for over a year, and my physical and mental health have improved more than I ever thought possible. The Klonopin I needed—to alleviate the panic attacks that were caused by the undiagnosed egg sensitivity—eventually worsened my mental health by creating dependence with inter-dose withdrawals and probably created most of my chronic fatigue syndrome symptoms.

HISTAMINE INTOLERANCE: FRESHNESS MATTERS

Like all Quiet Diets, Quiet Carnivore emphasizes fresh animal foods because aged, cured, and processed animal foods are high in histamine, a neurotransmitter that can cross into your bloodstream, attach to histamine receptors throughout your body, and cause any number of unpleasant symptoms.

From the moment a plant or animal dies, bacteria naturally present in the environment begin breaking down its proteins into histamine and other *biogenic amines* (protein fragments with biological activity). Histamine (and other amines, which we won't cover here) will continue to accumulate in food until you freeze it, boil it, or eat it, so the more "aged" foods are, the more histamine they will contain. Furthermore, we humans intentionally add bacteria to fresh foods to ferment them or cure them with salt to create products with longer shelf lives and more intense flavor profiles. We ferment milk to make cheese. We cure pork until it becomes bacon. We smoke salmon to make lox. We hang beef for months while a thick coat of bacteria breaks down its proteins to produce aged ribeye steaks. All of these "gourmet" foods can be extremely high in histamine.

The healthy intestine makes an enzyme that neutralizes histamine, so most people can tolerate reasonable quantities of high-histamine foods. However, if you have gut damage, take certain medications (including

“NSAIDs” like ibuprofen), drink alcohol, are under extreme stress, or are deficient in vitamin B6, vitamin C, copper, or zinc, your ability to neutralize histamine can be weakened, allowing histamine to breach your gut lining and run amok. This condition is called *histamine intolerance*, and it is fairly common, particularly in middle-aged women.

Histamine binds to four different receptors across eight different organ systems, so if a significant amount of histamine enters your circulation, it can cause a staggering variety of unpredictable symptoms throughout the body including migraines, daytime sleepiness, hives, stomach pain, uterine cramps, wheezing, palpitations, insomnia, itching, facial flushing, ankle swelling, racing/pounding heart, and blood pressure fluctuations.⁶

Unfortunately, there are no simple diagnostic tests for histamine intolerance, so if you suspect you may be dealing with this issue, the first step is to try a low-histamine diet (all three Quiet Diets are low in histamine). Because animal foods contain more protein than plant foods, they tend to accumulate more histamine as they age, but there are a few fresh plant foods that are naturally high in histamine or that stimulate our bodies to release histamine, such as avocado, spinach, and strawberries. The best way to reduce your histamine exposure is to buy the freshest meat, seafood, and poultry that you can find. This isn’t always easy, especially in the case of beef, because it is standard practice to age beef for several weeks to tenderize its fibers before it is distributed to grocery stores and butcher shops.

HISTAMINE INTOLERANCE TIPS

- Ask your grocer or local butcher if information about their receiving and packing schedules is available.
- Buy “frozen-at-sea” seafood or buy seafood from a fish market you trust.
- Check “packed on” dates on packaged meats before you buy, and cook or freeze foods the same day. Boiling or freezing will halt histamine production but cannot destroy any of the histamine that has already been created, and as

soon as you thaw a frozen food, histamine production will resume.

- Don't keep leftovers for more than 48 hours, because histamine continues to accumulate even in refrigerated foods. Cold cuts and rotisserie chickens can be particularly high in histamine.
 - Taking a histamine neutralizing enzyme supplement (*diamine oxidase* or DAO for short) before meals may help. See appendix B for more histamine intolerance resources.
-

QUIET CARNIVORE MACROS

Protein requirements may not be any higher on carnivore diets, but many people can eat more protein than on a standard ketogenic diet and still maintain therapeutic ketone levels. So instead of starting with 0.6 grams of protein per pound of ideal body weight (IBW) per day, start at about **0.8 grams of protein per pound IBW**. For example, if your IBW is 120 pounds, start with about 100 grams of protein per day. For reference: one pound of raw “80% lean” ground beef contains about 80 grams of protein, so if you were eating only ground beef, you’d need about 1.25 pounds (20 ounces) per day to meet your protein requirement. Carnivore diets contain no visible carbohydrate, so your “ketone control knob” on carnivore won’t be protein + carbohydrate, it will be protein alone. As protein intake goes up, so does your insulin level, bringing ketones down. If your ketones are too low or you’re not feeling well, try adding more fat to your meals. If your ketones are too high, increase your protein intake to 1.0 or 1.2 grams per pound IBW. If your ketones are running low and you’re not experiencing the mental health benefits you were hoping for, you may want to try lowering your protein intake to 0.7 or 0.6 grams per pound IBW.

Fat requirements, as always, should be tailored to your energy (calorie) requirements, but you should aim for a fat-to-protein ratio (FPR) of *at least* 1:1, meaning at least one gram of fat for every gram of protein.

For example, if your IBW is 120 pounds, you’d start with 100 grams of protein per day plus *at least* 100 grams of fat per day. (The same 1.25

pounds of ground beef in the example above contains about 115 g of fat.)

Some people have better results when they aim for a slightly higher FPR of 1.5:1, as this is more ketogenic, and Drs. Toth and Clemens recommend an even higher FPR of 2:1, so experiment with different ratios to see what feels right to you. To achieve higher fat-to-protein ratios, you usually need to add some animal fat to the cuts of meat typically found in most grocery stores, as much of the visible fat has been trimmed to suit modern consumer tastes. It is difficult to estimate the amount of protein and fat in various animal foods because the fat-to-protein ratio varies depending on the cut of meat, how much fat was trimmed at the butcher, how it is cooked, and even what the animal was fed. However, most cuts of red meat with visible fat contain at least a 1:1 ratio of fat to protein, so either keep very lean meats such as white meat poultry, shrimp, and pork loin to a minimum, or add plenty of fat to them. If you have access to a good local butcher shop, ask if you can special-order fattier cuts of meat or higher-fat ground meat. If you don't have a good butcher shop that stocks local pastured meats from humanely-raised animals, consider purchasing shares of fresh or frozen meat from a local or online meat CSA (community-supported agriculture) program.

Within a few weeks, as your metabolism adapts to a ketogenic carnivore diet, your appetite will become more reliable, you'll get a feel for how much protein and fat you need, and your eating plan will become more instinctive. Many women settle in at between 1 and 1½ pounds of meat/seafood/poultry per day, and many men settle in at between 1½ and 2 pounds per day, but let your appetite be your guide.

QUIET CARNIVORE FOOD LIST

Meat

- Fresh or freshly-frozen meat, poultry, and seafood are all permitted. Meat, seafood, and poultry must be free of starchy coatings, thickened gravies, sweetened sauces, sweetened marinades, and sugary rubs.
- Be careful with aged and processed meats such as cold cuts, bacon, aged beef, jerky, cured sausages, and smoked salmon as they can be high in histamine.

- Fattier cuts of meat contain higher fat-to-protein ratios, so you can have a little more and still stay within your protein allowance, and their higher fat content makes them more satisfying and more ketogenic. Higher-fat choices include:
 - Pork belly, pork butt, pork shoulder
 - Duck breast (skin-on)
 - Beef short ribs, ribeye steak, ground beef (80% lean or less)
 - Lamb ribs, lamb chops, ground lamb
 - Chicken thighs (skin-on)
- Red meat, shellfish, liver, and fatty fish contain more nutrients than white meat chicken or turkey breast and lean white fish filets. I also recommend including some gently cooked liver in your carnivore diet. If you don't care for the taste of liver, there are creative ways to sneak it into your life, such as mixing small amounts of liver into ground meat before cooking.

Broth (from red meat, seafood, or poultry) made without vegetable ingredients or spices

Animal fat Non-dairy animal fats of all kinds are permitted: tallow, duck fat, goose fat, chicken fat, lard, etc. Avoid bacon fat as it is high in histamine.

Salt

Water or plain seltzer

No fruits, vegetables, herbs, spices, coffee, or tea.

CARNIVORE CHALLENGES

All special diets pose social challenges, but carnivore diets can be particularly difficult. When you eat only meat, you require larger portions of meat per meal, which may mean ordering two or three entrees at restaurants (which can be expensive) or asking for extra helpings when eating at friends' homes, which can be expensive for them and awkward for you. If you're being diligent, you'll also need to request that no seasonings, sauces, or marinades are used. Some people find the carnivore diet boring,

and Quiet Carnivore can be particularly challenging in this regard because it omits dairy and eggs, which are such versatile and convenient animal foods. However, many are surprised at how satisfying the diet is and how much they enjoy eating this way. You will have to experience it for yourself to see what you think.

If you follow a strict carnivore diet for long enough, and you stray from your plan, you may experience exaggerated food reactions, perhaps because the enzyme systems your body uses to process plant toxins have basically gone to sleep, or because your microbiome has changed in response to your carnivore diet and needs time to readjust. If this happens to you, you may want to consider including very small quantities of plant foods from my Quiet Diet food list such as herbs, berries, or fruit-based tea in your carnivore diet to keep your system on its toes. This “carnivore-ish” approach is easier to maintain long-term but may result in fewer benefits for some individuals.

BEYOND SIX WEEKS

If after four to six weeks on Quiet Carnivore you are happy with how you feel, you may decide to continue longer-term with Quiet Carnivore, or try expanding your diet one food group at a time to see where your safe outer limit lies (see [chapter 16](#)).

If even a Quiet Carnivore diet doesn’t agree with you, it may be that certain animal foods don’t agree with you. Perhaps you don’t tolerate seafood well (like eggs and dairy, fish and shellfish are also among the “big nine” common allergy and sensitivity culprits), in which case it’s worth excluding it for a week as an experiment. To make matters more complicated, the animal foods we eat now aren’t what they used to be. Like us, animals have been burdened with pesticides, heavy metals, antibiotics, environmental pollutants, stress, lack of sun, improper diet, and unnatural diets based on grains and vegetable oils. Sadly, the fat in most pork and chicken sold in the United States is very high in linoleic acid (see [chapter 10](#)). For this reason, I advise using tallow or duck fat rather than lard or schmalz (chicken fat) as your go-to animal fats. Do your best to find (and advocate for) animal foods and fats sourced from healthy, humanely raised animals that are fed a species-appropriate diet.

If Quiet Paleo, Quiet Keto, and Quiet Carnivore don't help you, your symptoms may be unrelated to your diet, in which case you may want to consider consulting with a psychiatrist who specializes in functional medicine or integrative psychiatry to explore other potential root causes of mental health conditions.

CHAPTER 20

You Can Do It! Practical Tips and FAQs

Scientific understanding is all well and good, but changing the way we eat is hard, so this chapter is here to make it a little easier. If you're worried that Quiet Diet protocols seem too restrictive to manage long-term, remember that all you're doing right now is conducting an experiment of at least six weeks to gather information about how food affects your mental health. It's too early to know where it might take you. Regardless of whether you ultimately decide to follow some version of a Quiet Diet long-term, I would still encourage you to take bold action on behalf of your mental health by replacing any harmful products that may be in your kitchen with healthy whole foods.

DETOXIFY YOUR KITCHEN

Remove sugar, vegetable oils, dairy products, cereals, grains, legumes, processed foods, alcohol, flours, and any products made with vegetable oils or refined carbohydrates such as sauces, dressings, marinades, and mayonnaise from your home, as these items are unhealthy for everyone.

Restock Your Home for Health and Happiness

- Fresh/frozen meat, seafood, poultry
- Eggs
- Fresh/frozen fruits and vegetables (consult the Quiet Diet food lists)
- Tuna, salmon, and other types of fish in cans, jars, or pouches (careful if you have histamine intolerance)
- Herbs, seasonings, natural extracts, tart vinegars, and flavored salts (consult Quiet Diet food lists)

- Extra virgin/unrefined/cold-pressed fruit oils: olive oil, avocado oil, palm fruit oil
- Non-dairy animal fats such as duck fat and tallow (rendered fat from beef/lamb/sheep). Firebrand Meats offers lard that is lower in linoleic acid than commercially available lard in the United States.
- Seltzer
- Herbal teas made from kinder, gentler ingredients such as fruits rather than pungent spices

Tip: If you live with people who eat differently, store your food in separate areas of the refrigerator and pantry and consider asking them to keep items that are most tempting to you out of sight.

MEAL TIMING AND INTERMITTENT FASTING

As described in [chapter 5](#), taking frequent, prolonged breaks from food processing switches your metabolism into fat-burning mode, allowing essential maintenance and healing to take place. Intermittent fasting is a powerful clinical tool, but when working with the short-term discovery strategies in this book, I would advise you to focus on your food plan first, because it's easier and more comfortable to eat less frequently *after* your glucose and insulin levels have quieted down. This is especially true following the keto-adaptation phase of a ketogenic (or carnivore) diet, because keto-adaptation trains your cells to easily access fat for energy between meals. People following standard high-carbohydrate diets often get hunger pangs when attempting intermittent fasting, but these tend not to occur if you are already fat-adapted.

If your meal timing doesn't correct itself within a few weeks, or you're looking for ways to augment your efforts, you can then introduce meal-timing techniques such as fasting for fourteen to sixteen hours every night by not eating after dinner and delaying your breakfast by two hours. If you incorporate intermittent fasting into your metabolic plan, remember to stay well-hydrated and supplement with electrolytes as needed. Simple intermittent fasting methods like this can help you in a variety of ways.

Fasting is the ultimate elimination diet. If your mental health symptoms

improve during fasting windows, it strongly suggests that your condition is rooted in problems with food choices and/or metabolism, which would be a very hopeful and empowering discovery. For example, if you tend to wake up in a good mood in the morning but your mood deteriorates over the course of the day, it may be a sign that your food plan is making you depressed.

Fasting drops insulin like a stone. Everything we eat raises insulin to some extent, so nothing lowers insulin more effectively than not eating at all. If you stray from your healthy eating plan and need to get your glucose and insulin levels back under control, fasting is the most efficient way to accomplish this.

This is another reason why I recommend cleaning up your diet first. Switching back and forth between a high-carbohydrate/high-insulin diet and fasting can cause dramatic ups and downs in your glucose and insulin levels that can be physically and psychologically uncomfortable. Fasting is much easier on a low-insulin Quiet Diet.

Fasting is ketogenic. If you're having trouble achieving good blood sugar or insulin control with dietary strategies alone, combining intermittent fasting with Quiet Paleo, Quiet Keto or Quiet Carnivore is a powerful way to multiply your efforts. If you prefer not to follow a ketogenic diet, intermittent fasting may allow you to reap some of the metabolic benefits of ketosis without having to restrict carbohydrate intake.

Fasting fosters mindfulness. If the idea of not eating between meals or going for fourteen hours without food makes you anxious, observe your physical and psychological reactions without judgment as you practice these simple fasting techniques. Reassure yourself that it is perfectly safe to go without food for fourteen hours, even if it feels strange or uncomfortable. Exercises like this help deepen your understanding of your relationship with food.

Note: Please do not use fasting methods if you are underweight, have a history of anorexia or restrictive eating, or if you are pregnant or breastfeeding. Please do not fast for more than sixteen hours without consulting with a health care professional first. For more information about intermittent fasting, I recommend the work of fasting expert Dr. Jason Fung.

WHAT ABOUT SNACKING?

Quiet Diets don't include snacks because it's important to practice giving your body a break from food processing in between meals. However, it's normal to be hungry between meals when you're first transitioning to a ketogenic diet, so if you want to quell that feeling, it's okay to use whole-food ketogenic snacks for the first week or two until your appetite quiets down. Ideas include: a hard-boiled egg, cucumber or celery sticks with mashed avocado, a scoop of homemade chicken or tuna salad, or a handful of olives. If you're hungry in between meals even after the first couple of weeks, you may need to increase the amount of protein or fat in your meals.

WHAT ABOUT PALEO AND KETO CONVENIENCE FOODS?

You will be hard-pressed to find "paleo-friendly" and "keto-friendly" products that follow Quiet Diet principles, because the Quiet Diet consists of fresh whole foods, not of shelf-stable food products.

Most paleo convenience foods contain ingredients that *come from* whole foods but are actually refined, such as nut *flours* and coconut *sugar*, and a growing number of paleo snacks contain brain-risky cassava flour.

Many savory meat bars contain nightshade spices such as red pepper, and if you have histamine intolerance, shelf-stable meat products may not agree with you. If you do not have histamine intolerance, look for meat bars, dried sausages, and jerkies that are very low in carbohydrate and without nightshade spices. If you do discover that you tolerate nuts well, they make a very convenient whole-food snack, but be careful with nut *bars* held together with sticky sweet ingredients; check the label for carbohydrate content and stay within your safe limit.

Most "keto-friendly" products are neither keto nor friendly; these products are all very low in carbohydrate, but that's where the good news ends. Many are made with artificial sweeteners, vegetable oils, nut flours, and refined protein powders including whey protein powder, which spikes insulin almost as much as pure glucose does.

NATURAL AND ARTIFICIAL SWEETENERS

Honey is a paleo-friendly sweetener because it is all-natural and unrefined, but that doesn't mean it's a wise metabolic choice. Some varieties of honey do contain less glucose (and more fructose) than sugar, so they don't raise glucose levels as much as sugar does, but unfortunately, honey raises insulin levels *more* than sugar does.¹

If you're following a ketogenic diet, be careful with sweeteners. Sugar substitutes may keep your cravings for sweets alive, making it harder for you to stick to your healthy plan.

The better choices are allulose, erythritol, monk fruit, and stevia because they are extracted from natural sources, generally well tolerated, don't raise glucose levels, and have little to no effect on insulin levels. Avoid maltitol, xylitol, and sorbitol; these natural sugar alcohols cause blood sugar and insulin spikes as well as gastrointestinal distress, with maltitol being the worst offender. Avoid artificial sweeteners such as aspartame, Ace-K (acesulfame potassium), saccharine, and Splenda—another sweetener that can raise both glucose and insulin levels. (Splenda consists of sucralose mixed with dextrose and maltodextrin, two simple sugars that break down into glucose in the bloodstream.)

If you decide to include sweeteners on your ketogenic diet, use them sparingly and notice how they affect you. Unfortunately, scientific studies don't tell us what we really want to know about sweeteners: how they affect insulin and appetite levels in people following a ketogenic diet. Test your ketone levels before and one hour after drinking a beverage that contains your favorite sweetener. If your ketones drop significantly, the sweetener has probably raised your insulin level and should be avoided.

The good news is that interest in sweets usually diminishes substantially on a ketogenic diet, and many people discover an enhanced ability to appreciate the subtle sweetness in foods we don't typically think of as sweet, such as cucumbers, scallops, and broccoli. However, if you find yourself missing sweetness, here are some possibilities.

If you're on Quiet Keto, you could try a small portion of raspberries, blackberries, or strawberries from time to time. On Quiet Paleo, you can include more liberal amounts of fruit in your plan so long as your blood sugar stays in a healthy range. After you've completed the Quiet Diet protocol, if you find there are certain sugar-free treats you tolerate well and that don't trigger you to eat in an unhealthy way, you may decide to

incorporate them into your long-term plan, especially if they help you say no to clearly harmful alternatives made with sugar and flour. Choose low-carbohydrate treats with simple ingredients such as dairy-free dark chocolate sweetened with allulose, monkfruit, stevia, or erythritol instead of ultraprocessed snack bars containing whey protein, soy protein, or artificial sweeteners.

CAFFEINE

Caffeine is a psychoactive substance with complex effects on neurotransmitters, hormones, and metabolism. Caffeine stimulates the release of stress hormones (adrenaline and cortisol), and doses of 250 mg or more promote slightly higher glucose levels.² It also stimulates glutamate (the brain's "gas pedal") and dopamine activity, and suppresses the activity of GABA (the brain's calming neurotransmitter) and adenosine (a sleep-promoting neurotransmitter).³ These biochemical effects help to explain why caffeine can make you feel positive and alert but also why it is such a common cause of insomnia⁴ and panic attacks.⁵

If you consume caffeine in the form of coffee, keep in mind that coffee beans are seeds (see [chapter 12](#)). If you consume caffeine in the form of tea, be aware that tea contains tannins, which are antinutrients (see [chapter 13](#)).

It can take sixteen to twenty-four hours for caffeine to completely clear the bloodstream, so even a small quantity first thing in the morning can interfere with quality sleep in sensitive individuals. If you are a slow metabolizer, it can build up in your bloodstream over time and cause chronic anxiety and insomnia that may seem unrelated to the timing of caffeine intake. Caffeine destabilizes your appetite signaling system; it can suppress appetite in the short-term, but as it wears off later in the day, rebound hunger can occur.

Caffeine frequently leads to tolerance and physical dependence, meaning that you may require higher dosages over time to achieve the same stimulating effect, and that if you stop using it, you could experience withdrawal symptoms such as headaches, depression, fatigue, and difficulty concentrating. If you decide to try a caffeine elimination experiment (which I do recommend), cut back gradually to minimize withdrawal symptoms, and then give yourself at least thirty days to observe how you feel without

it.

WHAT ABOUT...

As you transition to a new style of eating, you will encounter challenges and obstacles, so here are some tips to help you navigate your way through these more easily.

Social Gatherings

When going to a dinner party, offer to bring a dish to share that is on your plan. Then there will be at least one dish there you can eat, and your host will feel less pressure to address your dietary needs. If you know the host will be serving a main dish with a sauce or topping you can't eat, ask if they might set aside a plain serving for you.

If you are going to a food-oriented public event where there won't be appropriate food, eat something before you go so you'll be less hungry and less tempted.

If people ask questions, find a neutral, self-focused way to explain why you are eating differently, such as "I'm trying an elimination diet experiment to help identify foods that might not agree with me." Phrasing like this keeps your mental and physical health concerns private and implies no judgment of their food choices.

Restaurants

Following Quiet Diet principles is challenging in restaurants, because most use vegetable oils and other low-quality ingredients. Most also pre-season their meat, seafood, and poultry with marinades and rubs or mix them with fillers, and then cover them in sauces, so even if you ask for the simplest dish on the menu and ask for it to be prepared without a sauce, you are very unlikely to be served something without unhealthy ingredients. That's okay, don't worry about trying to be perfect about your plan in restaurants (or anywhere else, for that matter)—just do the best you can. If you can manage to follow Quiet Diet guidelines even 90 percent of the time, you're doing great!

Traveling

Consider traveling with an insulated lunch bag and an ice pack. Cooler Shock ice packs are ideal because they contain a special gel that stays frozen for several hours and keeps foods ice-cold for up to ten hours. These are TSA-friendly when frozen solid, so you can take perishable food with you when flying out of U.S. airports.

Try to stay in hotel rooms with refrigerators or, better yet, choose a home stay with its own kitchen.

Cheat Days

The more closely you follow the Quiet Diet protocol, the more clarity you will gain and the more likely it will be that you will experience noticeable benefits. However, once you have completed the discovery process, whether or not you can safely “cheat” depends on your particular makeup and what your definition of cheating is. One person can enjoy some sweets or starchy foods and return easily to their healthy plan the next day, but another will be triggered to binge and may not return to their healthy plan for weeks or months. If you decide to take time away from your plan, I encourage you to draw the line at brain-healthy paleo. Splurging on bananas, salty nuts, and roasted sweet potatoes is much safer and healthier than crossing the line into the toxic realm of cookies, ice cream, and chips. It will also be easier to find your way back to your healthy plan afterward since whole foods are less addictive.

ISN'T EATING HEALTHIER MORE EXPENSIVE?

Eating healthy whole foods doesn't need to be more expensive and will usually save you money. Because of Western society's longstanding aversion to red meat, organ meats, and animal fat, some of the most nutritious animal foods are also some of the least expensive: dark meat poultry, higher-fat ground beef, eggs, mussels, liver, pork butt, etc. When you eat a lower-insulin diet, you're less hungry, you need less food, and you buy less food. You save lots of

money on snacks and sugary beverages. You will be healthier, so you can potentially save money on prescription medications, medical appointments, and medical procedures.

ADDICTIVE EATING

Academics continue to debate whether addiction to refined carbohydrates and ultraprocessed foods is a real phenomenon, but as a psychiatrist who has worked with thousands of patients over more than two decades (and as a human being who has personally struggled with this issue for five decades), I am convinced these addictions are real, that they are powerful, and that they represent a formidable threat to public health. I hesitate to refer to this growing scourge as “*food addiction*” because whole plant and animal foods don’t inspire destructive eating behaviors, whereas factory-made products have drug-like properties because they contain substances that have been extracted from foods, concentrated, and mixed together to create flavor, texture, and macronutrient profiles that most people find irresistible.

Addictions to these substances can be more difficult to manage than addictions to drugs or alcohol in many cases, because you can live and thrive without drugs and alcohol for the rest of your life, but you can’t live or thrive without eating regularly. Every time you eat, you are challenged to make a healthy choice, and that’s very difficult, particularly in our modern intoxicating food environment. It’s like asking a recovering alcoholic to walk into a bar whenever they feel thirsty and somehow summon the strength to ask for water each and every time. It’s not easy.

Being in ketosis is much like wearing a suit of armor that goes a long way toward protecting you against the temptations of our addictive food environment. When you’re burning fat for energy in between meals, your brain feels satisfied, so your appetite hormones won’t be pestering you for sugar. Most people find it much easier to turn down unhealthy foods when they’re in ketosis, *but ketosis is not a miracle cure*. Keto can curb cravings from the inside, but cravings can still be triggered from the outside: images, aromas, social situations, stress, etc. can all awaken the beast. Most addictive eaters have to battle a *lifelong* psychological vulnerability to

overeating, no matter what diet we follow. If you identify as an addictive eater, I've placed resources in appendix B to get you started on your recovery journey, but here are a few tips:

- Make a shopping list before going to the store and stick to it.
- Don't shop when you're hungry.
- Stay away from convenience stores.
- Consider asking someone else to do your grocery shopping or use a grocery delivery service, especially when you are feeling particularly vulnerable.
- Connect with others who are dealing with the same issue so you can support and learn from each other.
- Identify the products that you find most difficult to resist and redesign your daily patterns to avoid them as much as possible.
- Notice activities and situations that take your mind off of food and incorporate more of those into your life.

EXERCISE

Exercise is one of the pillars of excellent metabolic health. While it's true you can't out-exercise a bad diet, it's also true that you can't achieve optimal metabolic health through diet alone. Exercise works wonders for mood, energy, sleep, and metabolism. Exercise improves carbohydrate tolerance, brain blood flow, metabolic flexibility, and insulin sensitivity, and builds confidence, stamina, vitality, and strength. A key difference between plants and animals that we didn't cover in this book is that animals are designed to move. Don't be a plant! Find activities you enjoy and get moving. All types of exercise are healthy, but strength training and high-intensity interval training are more metabolically stimulating and time-efficient than prolonged, repetitive aerobic exercises such as running and biking.

Remember to take it easy with exercise during the early phase of keto-adaptation; let your energy level be your guide. If you are an athlete hoping to maintain your current activity level while adapting to a ketogenic or

carnivore diet, you may need to make adjustments to the plans in this book to support your performance goals. See appendix B for resources.

STAYING THE COURSE

Anything worth accomplishing takes effort, and you are worth the effort. You must invest in yourself to see change. Striving, learning, and improving as you reach new goals builds confidence, character, and self-respect. *Behind every no is a yes.* When you say no to cake, French fries, and candy bars, what are you saying yes to instead? Remind yourself of the goals you set out to accomplish and why they are important to you. Yes, there is some sacrifice involved in this journey, but the rewards can be nothing short of life changing. Mourn the loss of the “foods” that have caused you the most suffering and celebrate the benefits you are enjoying as a result of letting them go.

WISHING YOU GOOD MENTAL HEALTH

I’m excited for you to discover what is possible as you begin exploring the powerful connection between how you eat and how you feel. Maybe it’s not all about your genes, your childhood, your trauma history, your stress level, or some mysterious chemical imbalance in your brain that is making you depressed, anxious, unstable, unfocused, or forgetful. Maybe, at least in part, your mental health concerns have a potentially reversible metabolic and nutritional explanation. The best way to determine if this is the case is to change your diet and see if it changes your mind.

CHAPTER 21

Meal Plans and Recipes

Patricia Daly (<https://patriciadaly.com>) is an internationally recognized nutrition therapist who specializes in the practical implementation of metabolic health protocols and who credits the ketogenic diet for helping to quiet an aggressive case of eye cancer she was diagnosed with fifteen years ago. She is the co-author of *The Ketogenic Kitchen*, a cookbook and guide specifically for people with cancer. Born in Switzerland and living in Ireland, her creative recipes have a distinctive European flair.

Patricia transformed the Quiet Diet brain food rules into delicious recipes and seven-day meal plans to give you a taste of what it's like to eat in a quieter way. You can follow the meal plans as they're written, mix and match recipes you are most interested in, or use the Quiet Diet food lists to create your own recipes. Since many typical breakfast foods such as cereal, toast, and yogurt are excluded from all Quiet Diet food lists (and even eggs are excluded from Quiet Carnivore), some of the breakfast recipes in this book may seem unusual. However, if you are following Quiet Paleo or Quiet Keto, you can simply eat an omelette every morning if you prefer. Patricia chose the types of fat that best complimented each meal, but you're welcome to substitute any healthy fat of your choice so long as it is on the food list for the plan you are following.

Meal plans are based on a 2,000 calorie per day diet and provide approximately 75 grams of protein per day. Since your protein needs may be different, each recipe includes a field labeled "customize your protein" that helps you easily dial the protein content up or down, depending on your individual needs. (To estimate your daily protein requirements, see [chapter 17](#).) If you're following the meal plans, pay attention to the "plan ahead" notes that prompt you to prepare certain ingredients for the next day's recipes.

The Quiet Keto plans in this book contain about 75 grams of protein and 20 grams of net carbohydrate (total carbohydrate minus fiber) per day, making this a *modified ketogenic diet* of approximately 15 percent protein, 5 percent carbohydrate, and 80 percent fat.

Bon appétit!

QUIET PALEO MEAL PLAN

	Day 1	Page #
Breakfast	Veggie Omelette	here
Lunch	Shrimp Stir-Fry with Mayo	here
Dinner	Lemony Chicken with Green Olives and Mixed Leafy Salad	here
Plan ahead	Cook sweet potato for day 2 lunch	
Day 2		
Breakfast	Simple Meatballs with Mango Salsa	here
Lunch	Dilly Tuna Salad	here
Dinner	Pressure-Cooked Lamb Shoulder	here
Plan ahead	Cook potatoes for day 3 lunch	
Day 3		
Breakfast	Duck Breast in Savory Broth	here
Lunch	Egg Salad	here
Dinner	Roast Chicken	here
Plan ahead	Cook celery root and save chicken thighs for day 4 lunch	
Day 4		
Breakfast	Pan-Seared Pork Chops	here
Lunch	Chicken Wraps with Celery Root and Apple Salad	here
Dinner	Spaghetti Bolognese	here
Day 5		

Breakfast	Frittata Primavera	here
Lunch	Herb Pizza with Chermoula	here
Dinner	Ultramoist Salmon Parcels	here
Plan ahead	Make extra salmon for day 6 lunch	

Day 6

Breakfast	Chick-adoo Breakfast	here
Lunch	Creamy Salmon Vegetable Soup	here
Dinner	Roasted Pork Belly with Stir-Fry	here
Plan ahead	Cook the sweet potato for day 7 lunch	

Day 7

Breakfast	Tweaked Kedgeree	here
Lunch	Salmon-Stuffed Portobellos	here
Dinner	Liver Cakes with Pomegranate, Mint, and Fennel Salad	here

QUIET KETO MEAL PLAN

	Day 1	Page #
Breakfast	Veggie Omelette	here
Lunch	Shrimp Stir-Fry with Mayo	here
Dinner	Lemony Chicken with Green Olives and Mixed Leafy Salad	here
Plan ahead	Cook zucchini for day 2 lunch	

Day 2

Breakfast	Simple Meatballs	here
Lunch	Dilly Tuna Salad	here
Dinner	Pressure-Cooked Lamb Shoulder	here

Day 3

Breakfast	Duck Breast in Savory Broth	here
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Lunch	Egg Salad	here
Dinner	Roast Chicken	here
Plan ahead	Save chicken thighs for day 4 lunch	

Day 4

Breakfast	Pan-Seared Pork Chops	here
Lunch	Chicken Wraps with Artichoke Tapenade	here
Dinner	Spaghetti Bolognese	here

Day 5

Breakfast	Frittata Primavera	here
Lunch	Herb Pizza with Chermoula	here
Dinner	Ultramoist Salmon Parcels	here
Plan ahead	Make extra salmon parcels for day 6 lunch	

Day 6

Breakfast	Chick-adoo Breakfast	here
Lunch	Creamy Salmon Vegetable Soup	here
Dinner	Roasted Pork Belly with Stir-Fry	here

Day 7

Breakfast	Tweaked Kedgeree	here
Lunch	Salmon-Stuffed Portobellos	here
Dinner	Liver Cakes with Mint and Fennel Salad	here

QUIET CARNIVORE MEAL PLAN

	Day 1	Page #
Breakfast	Beefy Lamb Burgers	here
Lunch	Shrimp in Smashing Broth	here

Dinner	Chicken Thighs	here
Day 2		
Breakfast	Simplest Meatballs	here
Lunch	Tuna with Creamy Marrow Sauce	here
Dinner	Pressure-Cooked Lamb Shoulder	here
Plan Ahead	Save leftover lamb for day 3 lunch	
Day 3		
Breakfast	Crispy Duck Breast	here
Lunch	(lamb leftovers)	
Dinner	Roast Chicken	here
Plan ahead	Save chicken thighs for day 4 lunch	
Day 4		
Breakfast	Pan-Seared Pork Chops	here
Lunch	Shredded Chicken	here
Dinner	Beefy Lamb Burgers	here
Day 5		
Breakfast	Chicken Liver with Bacon	here
Lunch	Lickety Split Lamb Stir-Fry	here
Dinner	Ultramoist Salmon Parcels	here
Plan ahead	Marinate the chicken wings for day 6 breakfast and dry the pork belly for day 6 dinner	
Day 6		
Breakfast	Pan-Fried Chicken Wings	here
Lunch	Ribeye!	here
Dinner	Crackling-Top Pork Belly	here

Plan ahead	Save rendered pork fat for day 7 breakfast	
Day 7		
Breakfast	Scallops in Broth	here
Lunch	Salmon Muffins	here
Dinner	Lamb and Liver Stir-Fry	here

BREAKFASTS

Veggie Omelette

Makes 1 serving

2 tbsp duck fat
 1 small leek (3 oz), thinly sliced
 ½ cup celery root, grated
 2 portobello mushrooms, sliced
 3 eggs
 1 tsp dried thyme
 Salt and pepper, to taste
 ½ avocado, sliced

DIRECTIONS:

1. Heat the duck fat in a frying pan over medium heat.
2. Fry the leek, celery root, and mushrooms for 5–10 minutes or until softened.
3. Whisk together eggs and thyme, add salt and pepper to taste.
4. Pour the egg mixture over the vegetables, cover, and gently cook for 5–7 minutes or until the egg is set.
5. Garnish with sliced avocado and serve.

Nutrition facts per one serving: 54g fat, 27g protein, 28g total carbs, 8g fiber, 680 calories, 72% fat, 16% protein, 12% carbs

Keto variation:

Omit the celery root.

Nutrition facts per one serving: 54g fat, 26g protein, 13g total carbs, 7g fiber, 610 calories, 80% fat, 17% protein, 3% carbs

Customize your protein: 1 egg = 6 grams protein

Simple Meatballs with Mango Salsa

Makes 1 serving

2 tbsp tallow
1 spear broccoli (2 oz), finely chopped
1 small onion (2 oz), finely chopped
5 oz grass-fed ground beef
1 tsp ground oregano
½ tsp salt
ground black pepper, to taste

DIRECTIONS:

1. Melt tallow in a frying pan, cook the broccoli and onion over medium heat until soft, about 3 minutes.
2. Transfer vegetables to a mixing bowl (leaving some tallow in the pan), add remaining ingredients to the bowl, and mix together well with your hands.
3. Shape 4-6 meatballs (the smaller, the quicker they'll cook) and fry them over medium heat. Cover, but turn them occasionally, for about 6 minutes until cooked through.
4. Serve with mango salsa (recipe below).

Mango Salsa

½ cup mango, cubed (3 oz)
1 heaping tbsp cilantro leaves, finely chopped
1 tbsp lime juice

DIRECTIONS:

Mix the mango with chopped cilantro and lime juice.

Nutrition facts per one serving: 56g fat, 29g protein, 24g total carbs, 5g fiber, 700 calories, 71% fat, 17% protein, 12% carbs

Keto variation: Simple Meatballs

Serve without the mango salsa. Use mayonnaise (see recipe [here](#)) or mustard as dressing, if desired.

Nutrition facts per one serving: 55g fat, 27g protein, 9g total carbs, 3g fiber, 640 calories, 78% fat, 17% protein, 5% carbs

Carnivore variation: Simplest Meatballs

2 tbsp tallow
9 oz ground beef
Salt and pepper, to taste

DIRECTIONS:

1. Shape 8 meatballs and fry them in the tallow over medium heat.
2. Cover, but turn them occasionally, for about 6 minutes until cooked through.

Nutrition facts per one serving: 53g fat, 52g protein, 0g total carbs, 0g fiber, 690 calories, 70% fat, 30% protein, 0% carbs

Customize your protein: 1 oz ground beef = 5 grams protein

Duck Breast in Savory Broth

Makes 2 servings

4 tbsp duck fat
1 large duck breast (8 oz)
2 cloves garlic, minced
1 small sweet potato (4 oz), chopped
2 florets cauliflower (1 oz), chopped
1 cup bone broth
1 cup spinach leaves
1 avocado, sliced
Salt and pepper, to taste

DIRECTIONS:

1. Melt the duck fat in a frying pan. Cook the duck breast skin side down over medium heat to render the fat.
2. When the skin is crisp and brown, remove the duck from the pan and put it on a side plate.
3. Briefly blitz the sweet potato, cauliflower, and garlic with the bone broth in a food processor until smooth.*
4. Add the sauce to deglaze the frying pan of all the juices (stand back for this if the pan is hot). Turn the heat down, add the spinach, and let it wilt.
5. Add the duck breast back to cook it until done to your preference (an internal temperature of 130°F for medium rare, 145°F for medium).
6. Cut the duck breast in half, pour the vegetable broth over it, and serve with avocado slices.

* *If you prefer a thicker sauce than broth, start with ½ cup of bone broth and add more until you reach your desired thickness. Reducing bone broth will slightly reduce the total protein.*

Nutrition facts per one serving: 53g fat, 25g protein, 25g total carbs, 8g fiber, 630 calories, 75% fat, 16% protein, 9% carbs

Keto variation:

Omit the sweet potato.

Nutrition facts per one serving: 53g fat, 24g protein, 14g total carbs, 6g fiber, 590 calories, 81% fat, 16% protein, 3% carbs

Carnivore variation: Crispy Duck Breast

Makes 1 serving

1 large duck breast (8 oz)

Salt, to taste

1 tbsp duck fat

DIRECTIONS:

1. Pat the breast dry with a paper towel, score the skin (not the meat) in a cross-hatch pattern (this will make the skin crispier), and season with salt.
2. Melt the duck fat in a frying pan.
3. Lay the breast skin side down and cook over medium heat until the skin is crisp and brown (75% of cooking time should be on the skin side).
4. Flip the breast and cook the meat to your preference (an internal temperature of 130°F for medium rare, 145°F for medium).
5. Remove the duck from the pan and serve.

Nutrition facts per one serving: 43g fat, 34g protein, 0g total carbs, 0g fiber, 520 calories, 74% fat, 26% protein, 0% carbs

Customize your protein: 1 oz duck breast = 5 grams protein

Pan-Seared Pork Chops

Makes 2 servings

2 pork chops (go for the slightly thinner version, about 5 oz each)
6 tbsp lard

Salt and pepper, to taste
2 large spring onions or scallions (includes tops and bulb),
chopped
2 cups shiitake mushrooms (6 oz), sliced
6 sprigs fresh thyme
1 tbsp fresh rosemary
 $\frac{1}{2}$ cup bone broth
4 raw figs, washed and quartered

DIRECTIONS:

1. Time permitting, allow the pork chops to come to room temperature for 20–30 minutes before cooking. Pat dry with a paper towel.
2. Heat 3 tablespoons of the lard in a frying pan and sear the pork chops for 3–4 minutes per side until well browned. Season lightly with salt and pepper, transfer the chops to a plate, and cover with foil to keep warm.
3. Add the spring onions, mushrooms, and herbs to the frying pan with the pan juices and stir fry for about 5 minutes.
4. Pour in the bone broth to deglaze the pan, add the figs, and bring to a boil. Turn down the heat and stir in the rest of the lard. Simmer for a few more minutes.
5. Place the pork chops back in the pan. Cover and simmer another 2 minutes. Serve with more herbs if desired.

Nutrition facts per one serving: 62g fat, 45g protein, 24g total carbs, 5g fiber, 830 calories, 67% fat, 22% protein, 11% carbs

Keto variation:

Reduce the shiitake mushrooms to 1 cup and omit the figs.

Nutrition facts per one serving: 63g fat, 43g protein, 5g total carbs, 2g fiber, 760 calories, 74% fat, 23% protein, 3% carbs

Carnivore variation:

Makes 1 serving

1 pork chop (6 oz)
1 tbsp lard
Salt, to taste
 $\frac{1}{4}$ cup bone broth

DIRECTIONS:

1. Time permitting, allow the pork chop to come to room temperature for 20–30 minutes before cooking. Pat dry with a paper towel.
2. Heat the lard in a frying pan and sear the pork chop for about 4 minutes per side until well browned. Remove the chop from the pan, slice it into bite-size pieces, and season lightly with salt before placing it aside in a soup dish.
3. Pour in the bone broth to deglaze the pan and bring to a boil. Pour over the pork chop and serve.

Nutrition facts per one serving: 53g fat, 49g protein, 0g total carbs, 0g fiber, 690 calories, 70% fat, 30% protein, 0% carbs

Customize your protein: 1 oz pork chop = 8 grams protein

Frittata Primavera

Makes 1 serving

1 large spring onion (include tops and bulb), finely chopped
 $\frac{1}{2}$ cup butternut squash, grated or cubed into small pieces
5 asparagus spears, raw, chopped
2 tbsp olive oil
4 eggs
4 Kalamata olives, halved
1 clove garlic, minced
1 tbsp dried rosemary
Salt and pepper, to taste

DIRECTIONS:

1. Cook onion, squash, and asparagus in olive oil over medium heat. Cover pan, turn down the heat a bit, and stir occasionally while vegetables soften.
2. Beat the eggs in a bowl and fold in the olives, garlic, rosemary, salt, and pepper.
3. After about 5 minutes, or when the vegetables have softened a bit, add the egg mixture and cook everything on low to medium heat for about 10 minutes until the eggs are set. The frittata may be finished in the oven if you prefer a golden brown crust.

Nutrition facts per one serving: 54g fat, 30g protein, 24g total carbs, 6g fiber, 680 calories, 71% fat, 18% protein, 11% carbs

Keto variation:

Omit the butternut squash.

Nutrition facts per one serving: 54g fat, 29g protein, 11g total carbs, 4g fiber, 630 calories, 77% fat, 18% protein, 5% carbs

Customize your protein: 1 egg = 6 grams protein

Chicken Liver with Bacon (carnivore)

Makes 1 serving

2 tbsp lard
2 oz bone broth
5 oz chicken liver
3 oz uncured bacon (about 6 strips), cut into 1-inch strips
Salt

DIRECTIONS:

1. Melt the lard in a frying pan, add the bone broth, and bring to a boil.

2. Add the liver and bacon and cook on high temperature, but only for 2 minutes or less—until the meat has changed color.
3. Reduce the heat and simmer for a few more minutes—do not overcook to ensure the liver stays tender. Season well with salt and serve.

Nutrition facts per one serving: 62g fat, 55g protein, 0g total carbs, 0g fiber, 780 calories, 72% fat, 28% protein, 0% carbs

Customize your protein: 1 oz chicken liver = 5 grams protein

Customize your protein: 1 bacon strip = 5 grams protein

Chick-adoo Breakfast

Makes 1 serving

3 tbsp lard
½ cup bone broth
1 small chicken breast (3 oz)
1 cup Chinese cabbage (3 oz), thinly shredded
2 medium carrots, sliced into long strips with a julienne peeler
½ avocado, sliced
Salt and pepper, to taste
5 sprigs fresh cilantro, chopped

DIRECTIONS:

1. Bring the lard and bone broth to a boil in a frying pan.
2. Add chicken breast and cook in the boiling broth until the meat turns white.
3. Add the cabbage and carrots and simmer, covered, for 10–15 minutes or until the chicken is cooked through.
4. Top with sliced avocado, cover, and leave for 2–3 minutes.
5. Season with salt and pepper.
6. Garnish with chopped cilantro and serve.

Nutrition facts per one serving: 53g fat, 34g protein, 26g total carbs, 10g fiber, 680 calories, 70% fat, 20% protein, 10% carbs

Keto variation:

Omit the Chinese cabbage and carrots.

Nutrition facts per one serving: 53g fat, 28g protein, 12g total carbs, 5g fiber, 600 calories, 79% fat, 18% protein, 3% carbs

Customize your protein: 1 oz chicken breast = 8 grams protein

Pan-Fried Chicken Wings (carnivore)

Makes 1 serving

Plan ahead: For best results, rub the lard and salt into the wings, cover with a plate, and leave in the fridge overnight to marinate—the meat will become more tender.

2 tbsp lard

4 chicken wings (2 oz each)

½ cup bone broth

Salt, to taste

DIRECTIONS:

1. Heat the lard in a frying pan.
2. When the lard is hot, add the marinated chicken wings and sear them over high heat for about 2 minutes on both sides.
3. Reduce heat to low and continue cooking for 15 minutes or until the meat is tender. If you cover it, make sure to leave the lid cracked. Flip the wings about three times during cooking.
4. Take the frying pan off the stove top, remove the chicken, and add the bone broth to the pan. Deglaze to make sure to get all of the juices.
5. Serve the chicken wings and bone broth separately (the broth will make

the wings soggy).

Nutrition facts per one serving: 64g fat, 58g protein, 0g total carbs, 0g fiber, 820 calories, 70% fat, 30% protein, 0% carbs

Customize your protein: 1 chicken wing (2 oz) = 14 grams protein

Tweaked Kedgeree

Makes 2 servings

This is a heavily tweaked version of kedgeree, which is an ancient Indian dish that typically combines smoked fish, egg, onion, butter, and rice.

1 fillet of trout (6 oz)
6 tbsp duck fat
4 cups cauliflower, finely chopped
1 medium red onion (7 oz), finely chopped
2 cups mushrooms (6 oz), quartered
1 cup raw spinach
4 sprigs fresh cilantro, finely chopped
10 leaves fresh basil or lemon balm, finely chopped
Salt and pepper, to taste
4 hard-boiled eggs, quartered

DIRECTIONS:

1. Fry the trout in duck fat over medium heat for 3–4 minutes. Remove from the pan and cut it into 6 pieces. Keep the fish warm.
2. Finely chop the cauliflower or use your blender to make cauliflower rice. Add the onions, mushrooms, spinach, and cauliflower to the frying pan.
3. Toss in the herbs, season with salt and pepper, and cook about 5–10 minutes, stirring occasionally.
4. Add the trout pieces back in and stir-fry for another 5 minutes. Serve with the quartered eggs on top or mixed in—whichever you prefer.

Nutrition facts per one serving: 53g fat, 37g protein, 20g total carbs, 6g fiber, 680 calories, 69% fat, 22% protein, 9% carbs

Keto variation:

Swap the cauliflower for 3 cups broccoli (9 oz).

Omit the onion.

Nutrition facts per one serving: 53g fat, 37g protein, 13g total carbs, 5g fiber, 660 calories, 72% fat, 22% protein, 6% carbs

Customize your protein: 1 oz trout fillet = 6 grams protein

Scallops in Broth (carnivore)

Makes 1 serving

Most shellfish is very low in fat so be sure to add some rendered fat from another dish or a good amount of duck or other fat.

½ cup bone broth

5 tbsp rendered pork fat (saved from dinner on day 6) or other fat

10 oz scallops

Salt, to taste

DIRECTIONS:

1. Heat the bone broth and the rendered pork fat.
2. Add the scallops, bring to a quick boil, and poach over medium heat for about 4 minutes. If you prefer, you can also pan-fry the scallops in the pork fat to give them a golden crust.
3. Season well with salt and serve.

Nutrition facts per one serving: 63g fat, 52g protein, 0g total carbs, 0g fiber, 775 calories, 73% fat, 27% protein, 0% carbs

Customize your protein: 1 oz scallops = 5 grams protein

LUNCH

Shrimp Stir-Fry with Mayo

Makes 2 servings

2 tbsp olive oil
7 oz shrimp, peeled
2 cups broccoli (6 oz), chopped
3 medium carrots (6 oz), sliced into julienne strips
1 medium zucchini (7 oz), sliced into thin strips or grated
1 tsp salt
1/4 tsp ground black pepper
3 tbsp homemade mayo (recipe below)
1 avocado, mashed
4 large lettuce leaves
2 tbsp lemon juice

DIRECTIONS:

1. Heat olive oil in a frying pan over medium heat. Add shrimp and stir-fry for 3–4 minutes until they turn pink.
2. Add the broccoli, carrots, zucchini, salt, and pepper. Cover and simmer for 10 minutes until the vegetables are just softened.
3. Make the mayonnaise (see recipe below) and mix it with the mashed avocado.
4. Serve the stir-fry in lettuce leaves, topped with the mayo/avocado mash and lemon juice.

Homemade Mayonnaise

Makes about 1 cup

1/2 clove garlic, finely chopped

1 cup mild olive oil (organic)
2 tsp apple cider vinegar
1 very fresh egg
Salt and pepper, to taste

DIRECTIONS:

1. Sauté garlic in 1 tablespoon olive oil until soft. Let cool.
2. Place everything in a narrow glass jar (e.g. narrow-mouth mason jar) and let the oil rise to the top. Use egg straight out of the fridge.
3. Place stick/immersion blender at the bottom of the jar and turn on. Stay at the bottom of the jar for about 20 seconds until the mayo starts to emulsify. Then, slowly raise the blender out of the jar and pulse a few times until the whole mixture is thick and creamy.
4. Can be stored in the fridge for up to 3 days.

Nutrition facts per one serving: 57g fat, 27g protein, 31g total carbs, 12g fiber, 700 calories, 74% fat, 16% protein, 10% carbs

Keto variation:

Use only 1 cup of broccoli.

Omit the carrots.

Use 4 tbsp of mayonnaise.

Use only ½ avocado.

Nutrition facts per one serving: 59g fat, 25g protein, 13g total carbs, 5g fiber, 650 calories, 81% fat, 15% protein, 4% carbs

Customize your protein: 1 oz shrimp = 6 grams protein

Shrimp in Smashing Broth (carnivore)

Makes 1 serving

1/2 cup bone broth

3 tbsp duck fat
5 oz shrimp, peeled
3 oz salmon, cut into 3 pieces

DIRECTIONS:

1. Heat the broth in a shallow pan.
2. When hot, add the duck fat, shrimp, and salmon, and bring to a quick boil.
3. Simmer over medium heat until the shrimp and salmon are fully cooked.
Add salt to taste and serve.

Nutrition facts per one serving: 50g fat, 50g protein, 0g total carbs, 0g fiber, 640 calories, 70% fat, 30% protein, 0% carbs

Customize your protein: 1 oz shrimp = 6 grams protein; 1 oz salmon = 6 grams protein

Dilly Tuna Salad

Makes 2 servings

Plan ahead: Pre-cook the sweet potato (for the paleo recipe) or zucchini (for the keto variation) the night before and chill in the fridge.

1 medium sweet potato, (about 7 oz), cut into $\frac{1}{2}$ inch cubes
6 tbsp olive oil, divided
1 medium head lettuce, shredded
7 oz tuna in water, drained
2 tbsp capers
6 artichoke hearts, canned (drained and chopped)
2 tbsp apple cider vinegar
2 heaping tbsp homemade mayo (from [here](#))
10 sprigs dill, chopped
Salt and pepper, to taste

DIRECTIONS:

1. Toss the sweet potato with 1 tablespoon of olive oil and bake on parchment paper for about 15 minutes at 425°F, until tender. Let cool and chill in the fridge.
2. Mix lettuce, chilled sweet potato, and tuna in a bowl.
3. Sprinkle capers and chopped artichokes on top.
4. In a separate bowl, mix vinegar, mayonnaise, and 5 tablespoons olive oil; season with dill, salt, and pepper and pour over the salad.
5. Gently toss together and serve.

Nutrition facts per one serving: 59g fat, 28g protein, 25g total carbs, 5g fiber, 730 calories, 72% fat, 16% protein, 12% carbs

Keto variation:

Swap sweet potatoes for 1 medium zucchini (9 oz), sliced or cubed. Toss with 1 tablespoon olive oil and bake on parchment paper for about 5 minutes at 350°F until lightly softened, then chill.

Use 10 artichoke hearts.

Nutrition facts per one serving: 59g fat, 29g protein, 9g total carbs, 3g fiber, 670 calories, 79% fat, 17% protein, 4% carbs

Customize your protein: 1 oz canned tuna, drained = 7 grams protein

Tuna with Creamy Marrow Sauce (carnivore)

Makes 1 serving

¼ cup bone broth
2 oz bone marrow (e.g. beef)
Salt, to taste
7 oz tuna in water, drained

DIRECTIONS:

1. If you have bones with marrow, roast them at high heat for 10–15 minutes and then scoop the marrow out.
2. Heat the bone broth, add the marrow, and bring to a boil. Season well with salt.
3. In a blender, process the broth and marrow to get a creamy “sauce.” You can use more or less broth if you prefer a thinner or thicker sauce.
4. Add the tuna, heat for another 5 minutes or so and enjoy.

Nutrition facts per one serving: 54g fat, 59g protein, 0g total carbs, 0g fiber, 740 calories, 69% fat, 31% protein, 0% carbs

Customize your protein: 1 oz canned tuna = 7 grams protein

Egg Salad

Makes 2 servings

Plan ahead: Cook the potatoes the night before and chill in the fridge.

1 small red onion, very finely diced
1 tbsp avocado oil
6 eggs
4 heaping tbsp homemade mayo (see [here](#))
10 sprigs dill, finely chopped
Salt and pepper, to taste
2 stalks celery, finely chopped
1 cup cooked peeled potato, diced

DIRECTIONS:

1. Fry the onion in avocado oil until soft.
2. Bring eggs covered in cold water to a boil in a large saucepan; let simmer for 6 minutes, then carefully drain and place the pan in the sink.
3. Run cold water over the eggs for 1 minute, then refill the pan with cold

water and let it stand for about 10 minutes until the eggs have cooled down completely. Peel the shells off, carefully cut each egg into $\frac{1}{4}$ -inch pieces, and place them in a bowl.

4. Mix together the mayonnaise and dill and gently mix into the eggs. Fold the celery, potato, and red onion into the egg mixture. Season with salt and pepper.

Nutrition facts per one serving: 48g fat, 23g protein, 26g total carbs, 3g fiber, 620 calories, 70% fat, 15% protein, 15% carbs

Keto variation:

Replace potatoes with $\frac{1}{2}$ cup sliced cucumber.

Nutrition facts per one serving: 48g fat, 21g protein, 8g total carbs, 2g fiber, 540 calories, 80% fat, 26% protein, 4% carbs

Customize your protein: 1 egg = 6 grams protein

Chicken Wraps with Celery Root and Apple Salad

Makes 2 servings

Plan ahead: Save the chicken thighs from the roast chicken for this meal. Precook the celery root and chill.

1 tbsp duck fat
1 cup raw spinach
2 cooked chicken thighs, meat removed from the bone and sliced
6–8 large leaves of iceberg lettuce
1 cup sliced cucumber

DIRECTIONS:

1. Heat the duck fat in a frying pan, add the spinach and sliced chicken, stir-fry for 1–2 minutes until the chicken is slightly warm (if it's too hot, it might make the lettuce soggy).

2. Place the mixture onto the lettuce leaves.
3. Top with the sliced cucumber, wrap up, and enjoy.
4. Serve with the celery root and apple salad (recipe below).

Celery Root and Apple Salad

Makes 2 servings

1 cup celery root, peeled, cut into ½-inch cubes
5 tbsp olive oil, divided
15 sprigs fresh parsley, finely chopped
3 tbsp lemon juice
Salt and pepper, to taste
1 small apple, halved and thinly sliced

DIRECTIONS:

1. Toss the celery root with 1 tablespoon olive oil, and bake on parchment paper at 425°F for 10-15 minutes, or until tender. Cool and chill in fridge.
2. Mix 4 tablespoons olive oil, parsley, and lemon juice.
3. Season with salt and pepper and toss with the apple and celery root.

Nutrition facts per one serving: 58g fat, 32g protein, 17g total carbs, 4g fiber, 700 calories, 74% fat, 18% protein, 8% carbs

Keto variation: Chicken Wraps with Artichoke Tapenade

Substitute the artichoke tapenade for the celery root and apple salad.

Toward the end of heating the chicken and spinach, add tapenade to the mix.

Artichoke Tapenade

Makes 2 servings

3 tbsp olive oil
1 tbsp lemon juice
4 Kalamata olives
3 artichoke hearts, chopped
2 tsp herbes de Provence

DIRECTIONS:

1. Blend all the ingredients to a smooth texture (an immersion blender works best for this small quantity) and add salt if needed.

Nutrition facts per one serving: 56g fat, 31g protein, 10g total carbs, 5g fiber, 650 calories, 77% fat, 19% protein, 4% carbs

Carnivore variation: Shredded Chicken

Makes 1 serving

3 tbsp duck fat
2 boneless chicken thighs
Salt, to taste

DIRECTIONS:

1. Heat the duck fat in a frying pan. Use two forks or your hands to shred the leftover chicken from the bones. This will make it easier for the fat to be absorbed into the meat.
2. Reheat the meat while stirring for a few minutes, season with salt, and serve.

Nutrition facts per one serving: 60g fat, 53g protein, 0g total carbs, 0g fiber, 760 calories, 72% fat, 28% protein, 0% carbs

Herb Pizza with Chermoula

Makes 2 servings

9 oz ground lamb
2 cloves garlic, minced
2 sprigs fresh rosemary, finely chopped
Salt and pepper, to taste
1 small sweet potato (4 oz)
2 tbsp lard
1 small yellow onion, sliced (4 oz)
1 cup mushrooms (3 oz), sliced
1 serving chermoula (recipe below)

DIRECTIONS:

1. Preheat oven to 350°F.
2. Mix the ground lamb well with garlic, rosemary, salt, and pepper.
3. With your hands, press the mixture as thinly as you can onto a baking pan lined with parchment paper.
4. Bake for about 10 minutes until lightly brown.
5. Use a julienne peeler to cut the sweet potato into long strips. You can also just chop it finely.
6. Heat the lard in a frying pan and fry the onion, sweet potato, and mushrooms to your preferred doneness. Season well with salt and pepper.
7. When the meat base is cooked, spread 1 serving of chermoula onto it and top with the vegetables.

Chermoula

Makes 4 servings (use 1 serving for this recipe and store the rest in the fridge or freezer)

This sauce can be used to add flavor and nutrients to many different dishes. It's a very forgiving recipe—just tweak and modify until you find your “sweet spot.” It tastes best after the flavors have time to fuse.

One serving contains 200 calories and only 1 gram of net carbs—but 21

grams of healthy fat.

2 cloves garlic, chopped
6 tbsp olive oil, divided
½ bunch fresh cilantro leaves, chopped (1 oz)
½ bunch fresh parsley (1 oz), about 20 sprigs
¼ bunch fresh mint (1 oz)
1 tbsp lemon juice
1 tbsp lemon peel
Salt and pepper, to taste

DIRECTIONS:

1. Sauté the garlic in 1 teaspoon olive oil until soft. Let cool. Combine all ingredients into a blender and pureé. It will appear rather runny, but it becomes a firm paste once you put it in the fridge for a few minutes (depends on what you prefer).

Nutrition facts per one serving: 53g fat, 27g protein, 21g total carbs, 5g fiber, 660 calories, 72% fat, 17% protein, 11% carbs

Keto variation:

Increase mushrooms to 2 cups.

Swap the onion for 8 chopped artichokes (from 8 oz jar).

Omit the sweet potato.

Nutrition facts per one serving: 53g fat, 26g protein, 7g total carbs, 3g fiber, 600 calories, 80% fat, 18% protein, 2% carbs

Lickety Split Lamb Stir-Fry (carnivore)

Makes 1 serving

2 tbsp tallow
1oz bone broth
10oz lamb rump steak, sliced across the grain in ½-inch strips

Salt and pepper, to taste

DIRECTIONS:

1. Melt the tallow in a frying pan and add a splash of bone broth.
2. When the broth is boiling, add the lamb and stir-fry for 2-3 minutes or until it's cooked to your taste. Season well and serve.

Nutrition facts per one serving: 58g fat, 58g protein, 0g total carbs, 0g fiber, 750 calories, 72% fat, 28% protein, 0% carbs

Customize your protein: 1 oz lamb rump = 6 grams

Creamy Salmon Vegetable Soup

Makes 2 servings

Plan ahead: Make extra salmon parcels at dinner on day 5.

4 tbsp lard
1 small leek (3 oz), including the green part, roughly chopped
1 cup broccoli (3 oz), chopped
1 medium parsnip (6 oz), chopped
2 small zucchini (8 oz), chopped
2 cups bone broth
2 tsp salt
 $\frac{1}{2}$ tsp ground black pepper
2 medium salmon fillets, precooked
10 sprigs fresh cilantro leaves, chopped

DIRECTIONS:

1. Heat the lard over medium heat and gently fry the leek until softened, about 3 minutes.
2. Add the other vegetables and the bone broth. Season with salt and pepper.

3. Bring to a boil then immediately reduce heat. Simmer for 15 minutes. Blend the soup well in a food processor and return it to the pan.
4. Flake the salmon and add it to the soup. Cook for another 5 minutes to heat through, garnish with cilantro, and serve. If you prefer a very creamy texture, you can add the salmon in before blending.

Nutrition facts per one serving: 45g fat, 32g protein, 29g total carbs, 9g fiber, 630 calories, 63% fat, 22% protein, 15% carbs

Keto variation:

Replace the parsnip with 3 celery stalks.

Nutrition facts per one serving: 44g fat, 32g protein, 14g total carbs, 5g fiber, 560 calories, 71% fat, 23% protein, 6% carbs

Ribeye!

Makes 1 serving

1 ribeye steak, 1" thick (about 10 oz)

2 tbsp tallow

1–2 tsp salt

DIRECTIONS:

1. Bring the meat to room temperature—this will make all the difference!
2. Pat dry and rub the steak generously with 1 tablespoon tallow—this will form the amazing crust that we're looking for. You can also marinate the steak overnight. Season with salt.
3. Add the rest of the tallow to your skillet (a deep frying pan) and make sure it is smoking hot before putting in the steak. Stand back to avoid any splatter.
4. For a medium-rare steak, sear the meat for about 12 minutes, turning about 1 minute before the halfway point. You can use a meat thermometer (see details below) to cook the steak to your preference.

Pierce it into the side of the steak and monitor the temperature.

5. Take the steak off the stove before it reaches your desired internal temperature as it will continue to rise as it rests.
6. Rest your steak for 5 minutes before serving, covering lightly with parchment paper. This ensures the juices are reabsorbed back into the meat. This step is a “must” for any protein you cook hard and fast.

Doneness	Remove from heat at	After resting
Rare	118°F	120°F
Medium rare	125°F	130°F
Medium	136°F	140°F
Medium-well	143°F	150°F

Temperature guidelines (Source: <https://steakschool.com/learn/steak-temperature-chart/>)

Nutrition facts per one serving: 75g fat, 50g protein, 0g total carbs, 0g fiber, 875 calories, 77% fat, 23% protein, 0% carbs

Salmon-Stuffed Portobellos

Makes 2 servings

Plan ahead: Cook sweet potato the night before.

2 tbsp duck fat

2 cloves garlic, minced

4 large (or 6 smaller) portobello mushrooms (12 oz)

1 six-oz can red wild salmon, drained

1 small sweet potato (4 oz) (precooked and thickly sliced)

4 tbsp homemade mayo (see [here](#))

Salt and pepper, to taste

4 sprigs fresh dill, chopped

DIRECTIONS:

1. Heat the duck fat in a frying pan over medium heat, then add the garlic and mushrooms, top side down. Cover and gently cook for 5 minutes, stirring occasionally to avoid burning the garlic and to make sure the mushrooms don't stick to the bottom of the pan.
2. Flip the mushrooms and cook an additional 5 minutes.
3. Blend the salmon using a hand blender or food processor. Make sure there are no bones. Add the sweet potato and blend in.
4. Gently fold the mayonnaise into the salmon/sweet potato mixture and mix well. Season to taste.
5. Flip the mushrooms top-side down, fill with the salmon mix, and leave the filled mushrooms in the covered pan for another 5 to 10 minutes to heat the filling. They may also be heated in the oven if you prefer them hotter.
6. Decorate with the dill for color and serve.

Nutrition facts per one serving: 55g fat, 31g protein, 18g total carbs, 2g fiber, 680 calories, 72% fat, 18% protein, 10% carbs

Keto variation:

Swap the sweet potato for $\frac{1}{2}$ avocado (2.7 oz).

Nutrition facts per one serving: 60g fat, 31g protein, 12g total carbs, 3g fiber, 690 calories, 78% fat, 18% protein, 4% carbs

Customize your protein: 1 oz canned wild salmon = 6 grams protein

Salmon Muffins (carnivore)

Makes 1 serving

4 tbsp duck fat

1 6 oz-can wild salmon, in brine, drained

1 tsp salt

DIRECTIONS:

1. Preheat oven to 325°F.
2. Mix all ingredients in a blender until smooth.
3. Place the mixture in a silicone muffin pan and bake for a maximum of 20 minutes to avoid drying out.
4. Season with salt to taste and serve.

Nutrition facts per one serving: 63g fat, 60g protein, 0g total carbs, 0g fiber, 800 calories, 71% fat, 29% protein, 0% carbs

Customize your protein: 1 oz canned wild salmon = 6 grams protein

DINNERS

Lemony Chicken with Green Olives and Mixed Leafy Salad

Makes 4 servings

8 boneless chicken thighs, with skin (about 3 oz each, seasoned well with salt and pepper)
2 tbsp duck fat
2 medium onions, chopped or sliced
1 tsp salt
1/4 tsp ground black pepper
Pinch of saffron (optional)
1/2 cup bone broth
3/4 cup pitted olives
2 lemons, peeled and sliced
20 sprigs fresh chopped parsley
20 sprigs fresh chopped cilantro

DIRECTIONS:

1. Preheat oven to 350°F.

2. Use a covered pot that can be transferred from stove top to oven for this. Brown chicken skin-side down in duck fat over medium-high heat. Remove from the pan and set aside.
3. Reduce heat to medium, add onions, cover, and sweat for 5 minutes.
4. Add the salt, pepper, and saffron, stirring well, then return the chicken to the pot and add broth, olives, and lemons. Bring to a simmer, then cover and transfer to the oven for another 40 minutes.
5. Prepare the side salad and dressing (recipe below).
6. When the chicken is done, uncover and stir in parsley and cilantro. Spoon the sauce over the chicken to serve.

Mixed Leafy Salad with Vinaigrette

1 clove garlic, finely chopped
4 tbsp extra virgin olive oil, divided
2 tbsp apple cider vinegar
1 tbsp fresh lemon juice
1/4 tsp salt
Pinch of pepper
2 tsp thyme, fresh or dried
1 head of butterhead lettuce, washed and shredded
1/2 cup grated cooked beets
1 pomegranate, seeds only

DIRECTIONS:

1. Sauté garlic in 1 tablespoon olive oil until soft. Let cool.
2. Put remainder of olive oil, apple cider vinegar, lemon juice, garlic, salt, pepper, and thyme in a blender and process into a smooth vinaigrette.
3. In a large bowl, gently toss lettuce and beets.
4. Pour the vinaigrette over the vegetables, toss well, and top with pomegranate seeds before serving.

Nutrition facts per one serving: 50g fat, 26g protein, 23g total carbs, 6g fiber, 620 calories, 72% fat, 17% protein, 11% carbs

Keto variation (for salad only—chicken dish is the same):

Omit the beetroot and pomegranate.

Add ½ medium cucumber, sliced or cubed.

Add ½ avocado, cubed.

Nutrition facts per one serving: 52g fat, 25g protein, 13g total carbs, 5g fiber, 600 calories, 78% fat, 17% protein, 5% carbs

Customize your protein: 1 oz boneless chicken thigh = 8 grams protein

Chicken Thighs (carnivore)

Makes 1 serving

4 boneless chicken thighs (about 3oz each)
1 tbsp duck fat
½ cup bone broth
Salt and pepper, to taste

DIRECTIONS:

1. Preheat oven to 350°F.
2. You'll need a pot with a lid that you can transfer from the stove top to the oven. Brown the chicken thighs, skin side down, in the duck fat over medium-high heat.
3. Add the broth and bring to a simmer before transferring to the oven, lid on, for 40 minutes of roasting.

Nutrition facts per one serving: 69g fat, 51g protein, 0g total carbs, 0g fiber, 840 calories, 75% fat, 25% protein, 0% carbs

Customize your protein: 1 oz boneless chicken thigh = 8 grams protein

Pressure-Cooked Lamb Shoulder

Makes 4 servings

4 tsp coarse sea salt
2 cloves garlic, minced
 $\frac{1}{2}$ tsp ground black pepper
 $\frac{3}{4}$ cup lard, divided
1 lb lamb shoulder, cut into 1-inch dice
1 small red onion, sliced
2 tbsp fresh rosemary
2 tbsp dried sage or oregano
2 tsp dried thyme
2 cups organic bone broth
2 cups pumpkin, roughly chopped*
2 medium white potatoes (15 oz), peeled and quartered
2 cups mushrooms (6 oz), halved

DIRECTIONS:

1. Rub sea salt, garlic, and pepper into the meat—ideally 1 hour before cooking it.
2. Heat your pressure cooker to the “sauté” function and melt 3–4 tablespoons of the lard (or do this in a frying pan if your pressure cooker doesn’t have this function).
3. Brown the diced lamb for about 5 minutes, add onions and herbs, and stir for another minute.
4. Pour in 1 cup of bone broth and deglaze the bottom of the pot. Add the pumpkin and potatoes to the mix and simmer for 5 minutes (until you’re finished with step 5).
5. In a blender, process bone broth, mushrooms, and remaining lard until you have a smooth sauce, then add to pressure cooker.
6. Seal as per instructions and cook under pressure for 25 minutes. This recipe can also be made in a slow cooker on low for 8 hours to get the meat nice and tender.

* If pumpkin is not in season, you can substitute celery root, zucchini, or another type of squash of similar carbohydrate content.

Nutrition facts per one serving: 59g fat, 28g protein, 26g total carbs, 4g fiber, 740 calories, 72% fat, 15% protein, 13% carbs

Keto variation:

Omit the potatoes.

Nutrition facts per one serving: 59g fat, 26g protein, 8g total carbs, 2g fiber, 660 calories, 81% fat, 16% protein, 3% carbs

Carnivore variation:

Makes 2 servings

8 tsp coarse sea salt

4 tbsp lard

1 lb lamb shoulder, cut into 1-inch dice

2 cups bone broth

DIRECTIONS:

1. Rub plenty of coarse sea salt into the meat.
2. Heat your pressure cooker to the “sauté” function and melt the lard (you can use a frying pan if your pressure cooker doesn’t have this function).
3. Brown the diced lamb for about 5 minutes.
4. Pour in the bone broth and deglaze by making sure nothing sticks to the bottom of the pot.
5. Cover and seal as per instructions, then cook under pressure for 25 minutes. This recipe can also be made in a slow cooker on low, allowing 8 hours for the meat to become tender.
6. Serve the meat in the broth.

If you’re following the meal plan, save the second serving for lunch on day 3.

Nutrition facts per one serving: 66g fat, 48g protein, 0g total carbs, 0g fiber, 790 calories, 76% fat, 24% protein, 0% carbs

Customize your protein: 1 oz lamb shoulder = 5 grams protein

Roast Chicken

Makes 4 servings (and leftovers)

4 medium carrots, roughly chopped (8 oz)
1 large onion, quartered (9 oz)
2 cups Jerusalem artichokes, thickly sliced
4 cloves garlic, raw
10 sprigs fresh thyme
1 tbsp dried rosemary
1 whole chicken (4 lb)
1 lemon, halved
1 tsp salt, more to taste
½ tsp ground black pepper
1 tbsp red wine vinegar
½ cup bone broth

DIRECTIONS:

1. Preheat oven to 375°F.
2. Put the carrots, onions, Jerusalem artichokes, garlic, and herbs in the middle of a roasting pan. Place the chicken, breast side down, on top of the vegetables. Squeeze the lemon over the chicken, then put the lemon halves into the cavity of the chicken. Season with salt and pepper.
3. Cover the pan with parchment paper or aluminum foil and roast for 30 minutes, then remove the paper/foil and turn the oven down to 350°F.
4. Gently flip the chicken over, sprinkle with red wine vinegar, and pour the bone broth around the chicken and over the vegetables. Season again and cook for another 40 minutes.
5. Keep an eye on the vegetables so that they don't burn (you can remove

them early and keep in a covered pan to stay warm).

6. Turn off the oven, open the door, and let the chicken rest 10 minutes covered with parchment paper or aluminum foil. The legs should move about freely and when sliced between the leg and breast, the juices should run clear.
7. Remove the two thighs (leave the skin on) and save for lunch on day 4. Carve and remove the chicken breasts, remove the wings, and debone the rest of the meat (with the skin) for this dinner.
8. Serve the chicken and vegetables with some of the broth and rendered chicken fat from the baking pan.

Nutrition facts per one serving: 49g fat, 37g protein, 29g total carbs, 5g fiber, 700 calories, 63% fat, 21% protein, 16% carbs

Keto variation:

Replace the onion with 2 large, chopped spring onions (2 oz).

Replace Jerusalem artichokes with 2 halved bulbs of fennel, sliced (1 lb).

Nutrition facts per one serving: 46g fat, 32g protein, 17g total carbs, 7g fiber, 610 calories, 68% fat, 21% protein, 11% carbs

Customize your protein: 1 boneless chicken thigh (3 oz) = 20 grams protein; 1 chicken breast (5 oz) = 42 grams protein; 1 chicken wing (2 oz) = 14 grams protein

Carnivore variation: Roast Chicken

Makes 4 servings (and leftovers)

1 whole chicken (4 lb)

Salt, to taste

½ cup bone broth

DIRECTIONS:

1. Preheat oven to 375°F.

2. Place the chicken, breast side down, in a baking tray. Season generously with salt.
3. Cover the tray with parchment paper or aluminum foil and roast for about 30 minutes, then remove the paper and reduce the temperature to 350°F.
4. Gently tilt the baking tray and drain the rendered chicken fat into a glass jar. Flip the chicken over and pour the bone broth around the chicken. Season again and cook for another 40 minutes.
5. Turn off the oven, open the door, and let the chicken rest for 10 minutes while covered with parchment paper or aluminum foil. The legs should move about freely and if you slice between the leg and the breast, the juices should run clear.
6. Carefully remove the two thighs (leave the skin on) and save for lunch on day 4. Carve the chicken breasts, remove the wings, and debone the rest of the meat (with the skin).
7. Serve a 4 oz-piece of chicken breast alongside one of the wings. Carve them into slices and serve in the broth with the rendered chicken fat.

Nutrition facts per one serving: 56g fat, 52g protein, 0g total carbs, 0g fiber, 710 calories, 70% fat, 30% protein, 0% carbs

Spaghetti Bolognese

Makes 4 servings

Bolognese Sauce

4 tbsp olive oil
1 lb ground lamb
1 medium red onion, chopped (7 oz)
2 cloves garlic, minced
2 celery stalks, chopped
5 medium carrots, grated (10 oz)
1 tsp dried rosemary

1 tsp dried thyme
1 tsp dried oregano
Salt and pepper, to taste
2 cups mushrooms (6 oz), halved
1 small beet (3 oz), chopped
1 cup bone broth

DIRECTIONS:

1. Heat olive oil in a large frying pan over high heat. When hot, add the lamb and fry on high for 2–3 minutes.
2. Reduce heat to medium, add the onions, garlic, celery, carrots, herbs, and salt and pepper; cover pan.
3. Process the mushrooms, beet, and bone broth in a food processor until smooth, then add this sauce to the ground lamb and mix well.
4. Simmer the Bolognese sauce on medium-low heat for another 15–20 minutes. Season to taste with salt and pepper and serve with noodles (recipe below).

Noodles

4 medium zucchini (1 ¾ lb)
2 tbsp tallow
1 tbsp fresh lemon juice
Salt and pepper, to taste

DIRECTIONS:

1. Using a spiralizer or a julienne peeler, cut the zucchini into strips.
2. Heat the tallow gently in a large saucepan, then add the zucchini noodles and stir fry for about 10 minutes over medium heat. Squeeze the lemon juice over the noodles, season to taste, and serve with the Bolognese sauce.

Nutrition facts per one serving: 62g fat, 28g protein, 21g total carbs, 8g fiber, 730

calories, 76% fat, 16% protein, 8% carbs

Keto variation:

Use only 2 carrots in the Bolognese sauce.

Reduce the zucchini to 3 medium for the noodles.

Nutrition facts per one serving: 61g fat, 26g protein, 14g total carbs, 5g fiber, 690 calories, 79% fat, 15% protein, 6% carbs

Customize your protein: 1 oz ground lamb = 5 grams protein

Beefy Lamb Burgers (carnivore)

Makes 1 serving (2 patties)

These burgers can be made with all beef, all lamb, or a mixture of the two for a more complex flavor. If both meats are used, increase the serving size to two and double the tallow.

10 oz ground beef (20 percent fat)

—or—

9 oz ground lamb, (15 percent fat)

Kosher salt, to taste

1 tbsp tallow

DIRECTIONS:

1. Gently form two patties, taking care not to overwork the meat, which can lead to a tougher burger. Season liberally with salt.
2. Heat a cast iron pan on high heat until it just starts to smoke; add the tallow and the patties. Reduce heat to medium-high and let a crust form on the burger (about 3 minutes). Flip and finish cooking to your preferred doneness (see [here](#) for temperature guidelines).

Nutrition facts per one serving: 69g fat, 49g protein, 0g total carbs, 0g fiber, 820 calories, 76% fat, 24% protein, 0% carbs

Customize your protein: 1 oz ground beef or lamb = 5 grams protein

Ultramoist Salmon Parcels

Makes 4 servings

*½ small head cauliflower (10 oz)
4 small sweet potatoes
1 clove garlic, raw
½ cup duck fat
4 medium salmon fillets, with skin (5 oz each)
8 fresh basil leaves
12 fresh sprigs of cilantro
1 lime, quartered
2 tbsp lime juice
4 tbsp olive oil
2 tsp salt (or more to taste)
½ tsp ground black pepper*

DIRECTIONS:

1. Preheat oven to 375°F.
2. Chop the cauliflower and sweet potatoes into small chunks. Put them onto a baking sheet with the garlic, generously season with salt and pepper, and cover with duck fat. Bake for 20 minutes, stirring occasionally.
3. Put each salmon fillet onto a large rectangle of parchment paper. The salmon pieces should be long and skinny so you're able to fold them over. Stuff each fillet with a couple of basil leaves, three sprigs of cilantro, and a slice of lime.
4. Sprinkle each fillet with a little lime juice and one tablespoon of olive oil, and season well with salt and pepper. Wrap them up in parchment paper and tie loosely with string if needed.
5. Place the parcels in a baking pan, sprinkle them with 3–4 tablespoons

water, and bake for 10–15 minutes. If you'd like them to be “rare” in the middle, let them cook for about 10 minutes. If you prefer them well done, bake for 15–20 minutes. They will be tender and moist either way.

6. Serve a salmon parcel with the roasted vegetables.

Nutrition facts per one serving: 49g fat, 34g protein, 22g total carbs, 4g fiber, 650 calories, 67% fat, 21% protein, 12% carbs

Keto variation:

Increase the cauliflower to 1 small head (20 oz).

Swap the sweet potato for 3 cups of mushrooms.

Nutrition facts per one serving: 49g fat, 36g protein, 10g total carbs, 4g fiber, 610 calories, 72% fat, 24% protein, 4% carbs

Customize your protein: 1 oz salmon fillet = 6 grams protein

Carnivore variation:

Makes 1 serving

2 medium salmon fillets, with skin (5 oz each)

2 tbsp duck fat

1 tsp salt

DIRECTIONS:

1. Preheat oven to 375°F.
2. Put the salmon fillets onto a large rectangle of parchment paper. The salmon pieces should be long and skinny so you're able to fold them over.
3. Mix the duck fat and salt—if the duck fat is too solid, gently melt it in a pan.
4. Spread the fat evenly onto the two fillets. Wrap them up in the parchment paper and secure loosely with string if needed.

5. Place the parcels in a roasting pan, sprinkle with some water (3–4 tablespoons), and bake for 10–15 minutes before serving.

Nutrition facts per one serving: 61g fat, 58g protein, 0g total carbs, 0g fiber, 770 calories, 70% fat, 30% protein, 0% carbs

Roasted Pork Belly with Stir-Fry

Makes 4 servings

Pork belly is one of the fattiest cuts of meat and the protein content is much lower. You can modify this by using skinless pork belly (see protein calculations below).

Zest of 4 lemons

4 +1 cloves garlic, minced

A handful of fresh parsley, finely chopped

1½ lb pork belly, raw

Salt and pepper, to taste

1 small red onion (2 oz), chopped

5 cups Chinese cabbage (15 oz), finely shredded

1 large sweet potato (10 oz), diced into small cubes (about ½-inch)

1 tbsp fresh chives, finely chopped

1 tbsp dried thyme

DIRECTIONS:

1. Preheat oven to 450°F.
2. Blend lemon zest, 4 cloves of garlic, and parsley together to make a gremolata. Season the pork belly with salt and pepper, then cover with a layer of gremolata. Roll the belly up like a Swiss roll and tie it tightly with string in the middle and at the two ends to secure.
3. Roast for 45 minutes, then reduce heat to 275°F and roast for two more hours.
4. Forty minutes before the meat is done, transfer some of the rendered pork

fat to a frying pan and gently fry the onion and remaining clove of garlic. Add the cabbage and sweet potato to the pan, mix everything well, and cook for 30 minutes on low, covered, stirring occasionally.

5. Season with salt and pepper and add the herbs toward the end. Serve with a portion of the pork belly.

Nutrition facts per one serving: 79g fat, 17g protein, 20g total carbs, 4g fiber, 850 calories, 83% fat, 8% protein, 9% carbs

Keto variation:

Add an extra 2 cups of shredded cabbage (7 cups total).

Omit the sweet potato.

Nutrition facts per one serving: 794g fat, 16g protein, 7g total carbs, 3g fiber, 790 calories, 89% fat, 8% protein, 3% carbs

Customize your protein: 1 oz pork belly = 3 grams protein (15 grams fat);
3 oz skinless pork belly = 6 grams protein (3 grams fat)

Crackling-Top Pork Belly (carnivore)

Makes 4-5 servings

Plan ahead: Dry the pork belly with a paper towel, score the skin with a sharp knife, and leave uncovered in the fridge overnight to dry out the skin.

1½ lbs pork belly

2 tsp salt

½ cup bone broth

DIRECTIONS:

1. Preheat oven to 475°F.
2. Season the pork belly with plenty of salt, massaging it well into the meat.
3. Place the pork into a roasting pan, skin side up. Roast for 40–50 minutes,

checking occasionally after 30 minutes to ensure it doesn't get too brown. The crackling should be golden and super crunchy.

4. Reduce the oven to 300°F and place the pork directly on the top rack of the oven. Make sure you put an empty tray under the pork to catch the juices.
5. Cook the meat for another 2–4 hours. After 2 hours, the meat will be soft and easy to carve; after 4 hours, the meat will be soft enough for “pulled pork.”
6. To serve, heat the broth with some of the pork drippings to use as a dipping sauce.

If you're following the meal plan, be sure to set aside some of the rendered pork fat for breakfast on day 7.

Macros for this recipe are based on 4 oz pork belly with skin or 5 oz skinless pork belly.

Nutrition facts per one serving: 74g fat, 40g protein, 0g total carbs, 0g fiber, 830 calories, 80% fat, 20% protein, 0% carbs

Customize your protein: 3 oz pork belly = 8 grams protein (fat = 45 grams); 3 oz skinless pork belly = 17 grams protein (fat= 8 grams)

Liver Cakes with Pomegranate, Mint, and Fennel Salad

Makes 4 servings (about 12 liver “muffins”)

15 oz chicken liver
4 eggs
 $\frac{1}{2}$ cup duck fat (3 oz)
4 tbsp olive oil
1 medium apple (6 oz)
2 small onions (4 oz)
1 tsp salt
 $\frac{1}{4}$ tsp ground black pepper

1 tsp dried thyme

DIRECTIONS:

1. Preheat oven to 325°F. Put all ingredients into a strong blender and pulse until you have a smooth, fairly liquid paste.
2. Pour into a silicone 12-muffin pan and bake for 15–25 minutes. If you don't have a muffin pan, you can use a small baking dish lined with parchment paper. The mixture will be spread a bit thinner and will take less time to bake.
3. Invert the liver cakes onto a plate and serve with the Pomegranate, Mint, and Fennel Salad.

Pomegranate, Mint, and Fennel Salad

4 tbsp olive oil

2 bulbs fennel (6 oz), finely sliced across the width of the bulb

1 head of celery (about 7 stalks), finely chopped

12 heaped tbsp pomegranate seeds (about 2 small pomegranates [12 oz])

Salt and pepper, to taste

15 sprigs fresh mint, chopped

20 sprigs fresh tarragon, chopped

Juice of 1 lemon

DIRECTIONS:

1. Heat the olive oil in a frying pan and gently cook the fennel and celery for 15 minutes or until soft. Allow to cool.
2. Mix with the pomegranate seeds and herbs, then top the salad with the lemon juice and serve.

Nutrition facts per one serving: 59g fat, 28g protein, 22g total carbs, 7g fiber, 690 calories, 76% fat, 16% protein, 8% carbs

Keto variation:

For the liver cakes, swap the medium apple for a small one (3 oz).

For the salad, omit the pomegranate seeds and swap the lemon juice for 3 tablespoons apple cider vinegar.

Nutrition facts per one serving: 59g fat, 27g protein, 11g total carbs, 4g fiber, 660 calories, 81% fat, 16% protein, 3% carbs

Customize your protein: 1 oz chicken liver = 5 grams protein

Lamb and Liver Stir-Fry (carnivore)

Makes 1 serving

1 tbsp tallow
5 oz ground lamb
5 oz lamb, beef, or calf liver, sliced
1 tsp salt

DIRECTIONS:

1. Heat the tallow in a pan and fry the ground lamb over medium heat.
2. When it's browned, add the sliced liver and stir-fry for no more than 3–5 minutes. Do not overcook so that the liver stays nice and tender.
3. Season well with salt and serve.

Nutrition facts per one serving: 62g fat, 53g protein, 0g total carbs, 0g fiber, 770 calories, 72% fat, 28% protein, 0% carbs

Customize your protein: 1 oz ground lamb = 5 grams protein; 1 oz lamb liver = 6 grams protein

Slow-Cooked Chicken

Makes 4 servings (and leftovers)

Tossing a chicken in the slow cooker is a great trick for lightening the

cooking burden for the week. You can either enjoy it on its own or use it as the foundation for multiple meals, such as the Chick-adoo Breakfast, the Chicken Wraps, and the Shredded Chicken. You will also have plenty of lovely chicken stock that you can freeze in batches.

1 tbsp duck fat
1 whole chicken (4 lb)
1 tbsp dried rosemary
3 tsp salt
4 tbsp fresh rosemary

DIRECTIONS:

1. Heat the duck fat in the slow cooker and brown the chicken using the “fry” function. (If your slow cooker doesn’t have this feature, use a frying pan and then transfer the browned chicken to the slow cooker.) Season well with salt.
2. Cover the chicken with water and add the rosemary. Cover with the lid and cook overnight for at least 6–8 hours on low heat. Check that the chicken is fully cooked by wiggling the wing—it should be very loose. The chicken should be very tender and juicy—the flesh should easily come off the bones.

Nutrition facts per one serving: 56g fat, 52g protein, 0g total carbs, 0g fiber, 710 calories, 70% fat, 30% protein, 0% carbs

Acknowledgments

This book builds upon the ideas, talent, and hard work of so many others:

My intellectual heroes who think big thoughts, ask new questions, challenge pre-existing beliefs, do their own homework, and push knowledge forward. Thank you, Nina Teicholz, Amber O’Hearn, Dr. Ben Bikman, Gary Taubes, Dr. Jason Fung, Dave Feldman, Dr. Mike Eades, Belinda Fettke, Dr. Thomas Seyfried, Nicolette Hahn Niman, and Dr. Tim Noakes.

My clinician-researcher heroes who refuse to accept the limitations of conventional care; remain open-minded and curious; possess the humility to believe, respect, and learn from their patients; seek new ways to help them; and then do the hard work of analyzing and publishing their findings so that others may benefit. These include Dr. Sarah Hallberg, Dr. Jeff Volek, Dr. Eric Westman, and Dr. David Unwin.

My metabolic psychiatry heroes one and all, who are changing the way we think about, feel about, study, and treat mental illnesses. Thanks especially to Dr. Iain Campbell for contributing your personal and professional insights into bipolar disorder to [Chapter 9](#), to Dr. Ignacio Cuaranta for your steadfast friendship and for reminding me that pressure makes diamonds, and a special thank-you to Prof. Stephen Cunnane and Dr. Adrian Soto-Mota for their expert reviews of my biochemistry chapter. Thanks also to Bitten Jonsson, Heidi Giaevers, and Dr. Jen Unwin for teaching me and so many others about sugar and ultraprocessed food addiction. I am deeply grateful to Jan Ellison Baszucki for valuing and supporting not only my work, but the work of so many others in this exciting new field, and for knitting us together into a collaborative community. Thank you for spearheading the metabolic psychiatry revolution. We could not have dreamed up a more capable champion for the cause. I also have a special place in my heart for Dr. Albert Danan, whose

devotion to his patients accidentally culminated in the world's largest inpatient study of ketogenic diets for serious mental illness. Albert, it was an honor and a privilege to help bring your work to light.

My nutrition professional heroes—Independent thinkers who dared to break the mold and who skillfully teach people how to implement metabolically sound dietary strategies into their lives. Thank you Denise Potter, Franziska Spritzler, Dr. Adele Hite, Dr. Zoe Harcombe, Ulrike Gonder, Julia Tulipan, and Miriam Kalamian. I am particularly grateful to Patricia Daly for transmogrifying my unorthodox dietary philosophies into recipes and meal plans for this book and to Beth Zupec-Kania, expert among experts, for her careful review of chapters pertaining to ketogenic diet science and implementation.

Mission-driven conference organizers, community builders, and clinician influencers who have welcomed me onto their stages and virtual platforms, helping me grow an audience for the ideas that inspired this book, particularly Doug Reynolds, Pam Devine, Dr. Jeff Gerber, Dr. Rod Tayler, Dr. John Schoonbee, Dr. Ken Berry, Dr. David Perlmutter, Victoria Field, Dr. Angela Poff, and Dr. Dom D'Agostino. Special thanks to Josephine and Stephan Barbarino for magical conference experiences in the Swiss Alps and for going above and beyond in supporting this book project by hosting me as your first scientist-in-residence in Burghausen, Germany.

The people with lived experience who generously shared their personal stories for the book so that others may benefit from their journeys, including Karl Windinwood, Penny Figtree, Fran, Eric, Lisa, and others who chose to remain anonymous. Thanks also to all the patients I've had the privilege of working with over the past twenty-five years. You have been my greatest sources of inspiration and my greatest teachers.

The publishing professionals who gave of their time and talents to help make this book a reality, including Stuart Horwitz, and everyone on the staff at Hachette/GCP Balance. I am especially grateful to my literary agent Alex Glass and my editor Nana Twumasi for believing in this project from the beginning, helping me develop it for a general audience, and for patiently guiding me through the dizzying corn maze that is the commercial publishing process.

My cherished inner circle of friends, family, and colleagues who encouraged me, tolerated me, and helped me through what turned out to be

the most challenging professional task I have ever voluntarily undertaken. I apologize again for being so unavailable for so long and I know I have a lot of work to do to rebalance the scales. For time spent poring over chapter drafts, I am indebted to Terri Corona for laughing and gasping in all the right places, to Dr. Jayne Farley for pithy commentary, to Gloria Neggers for offering the valuable dual perspective of clinician and lay reader, and to Eileen Rakouskas for advocating for readers without a science background. Extra special thanks to you, Eileen, for providing invaluable logistical, emotional, and moral support for this project from start to finish—putting the paleo meal plans through their paces, running errands when deadlines were looming, reading (and re-reading) chapters for tone and clarity, and repeatedly making both of your ears available at a moment’s notice. Thank you to Christine Flannery for reminding me that some readers just want to be told what to do, and to Dr. Lieneke van der Griendt for hosting us at her home in the Netherlands while we finished the manuscript—truly gezellig. Thanks to Amber O’Hearn for her expert review of my carnivore diets chapter. Thanks also to Jen Unwin for lending eagle-eyed expertise to the opening chapters and serving up delicious vittles from her (hot hot hot!) Aga range cooker—and to David Unwin for punctuating our manuscript time with hunting owls, menacing cows, and lovely strolls through the re-wilded English countryside.

I’m grateful to Jennifer Bullock, PhD-to-Be, for her invaluable research assistance. Thanks also to Dr. Gayle Goren, Dr. Michaela Millott, and Dr. Jennifer Harris, who have all stuck by me since residency and provided wise counsel even as I have ventured beyond the boundaries of traditional psychiatric practice into the Wild West of nutritional and metabolic psychiatry.

Special thanks to my multi-talented sister, Cheri Ede for countless hours of deliciously expert recipe testing and recipe editing, as well as many other forms of assistance, both tangible and intangible, including recording and annotating chapters that needed special attention. Your support means the world to me. And deep gratitude to my mother, Mary Ede, who will be ninety years old when this book is published. An attentive professional caregiver and fierce advocate for adults with intellectual disabilities, it is her emotional intelligence, work ethic, kindness, creativity, generosity, and sense of humor that I aspire to embody in my own professional and

personal life. Thanks for believing in me, always being there for me, and for helping me not take myself too seriously. I am so proud to be your daughter.

And Suzi Smith, my partner in every sense of the word, for caring about, sacrificing for, and working on this book every bit as much as I did. Thank you for your boundless patience with my many shortcomings, your unwavering love and support, and for everything you've done to make everything I've done in the past ten years more visually engaging, easier to digest, and a lot more fun. As our friend Dr. Jen Unwin is fond of saying, "Everybody needs a Suzi."

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About the Author



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Dr. Georgia Ede is a Harvard-trained psychiatrist specializing in nutritional and metabolic psychiatry. Her pre-medical career was spent as a research assistant at the Joslin Diabetes Center in Boston, the Institute for Diabetes Research in Munich, and other academic laboratories in the fields of biochemistry, immunology, and metabolism. Her two decades of clinical psychiatry experience include twelve years as a college mental health specialist at Smith College and Harvard University Health Services, where she was the first to offer nutrition-based therapies as an alternative to psychiatric medication. Dr. Ede writes about food and the brain for *Psychology Today* and for her own website, DiagnosisDiet, and has been

speaking on the global stage about nutrition science, nutrition policy reform, and nutritional approaches to psychiatric conditions for more than a decade. In her Massachusetts-based private practice, she consults with patients and colleagues around the world about how to use ketogenic diets and other nutrition-based interventions to address the root causes of mental health conditions, often reducing the need for psychiatric medication. To improve patient access to metabolic psychiatry services, in 2020 she developed the first and only medically accredited Ketogenic Diets for Mental Health Clinician Training Program, in which she teaches practitioners how to safely use ketogenic therapies to treat mental health disorders. In 2022, she co-authored the first inpatient study of the ketogenic diet for serious mental illnesses and was honored to be named a recipient of the Baszucki Brain Research Fund's first annual Metabolic Mind Award.

Appendix A

Recommended Tests

Laboratory Tests

- **Fasting**¹ comprehensive metabolic panel (CMP), which includes a fasting glucose. The CMP includes tests of acid-base balance, electrolyte balance, kidney function, and liver function.
- **Fasting* lipid panel** (aka “cholesterol check”)
- **Hemoglobin A1C:** This is an estimate of your average blood glucose levels over the past three months.
- **Vitamin B12:** Levels below 220 pg/ml indicate deficiency. Levels above 500 pg/ml are ideal (note that this cutoff is much higher than the cutoff listed on standard lab reports). If your level falls between 220 and 500, your B12 status is questionable and needs further evaluation, in which case you should get a methylmalonic acid (MMA) level. A normal MMA (below 270 nmol/l) is reassuring that you have enough functional B12 in your system. An elevated MMA (above 370 nmol/l) suggests B12 deficiency. If your B12 level is low despite plenty of nutritious animal foods, consult with your health care practitioner. Many common health conditions and medications can interfere with B12 processing and absorption.
- **Serum ferritin:** This test can detect iron deficiency in its earliest stages, before it advances to anemia. Ferritin should be at least 100 ng/ml, a cutoff that differs from the normal range listed on standard lab reports.¹ If your level is below 100, you are running low on iron. A ferritin level two to three times the upper limit of normal is a common sign of insulin resistance,² and extremely high levels may indicate hemochromatosis (an iron storage disease).³

- **Thyroid function tests:** These may include TSH, T3, T4, free T3, free T4, anti-thyroid peroxidase antibody (anti-TPO), and anti-thyroglobulin antibody (TgAb) tests. These can be complicated to interpret, so if any of these are abnormal, consult with your physician. Note that levels of T3 (active thyroid hormone) tend to go down on ketogenic diets but this is unlikely to signal impaired thyroid function or impending hypothyroidism.⁴
- **C-reactive protein (CRP):** This is a test for inflammation. Below 1.0 mg/ml is ideal.
- **Complete blood count (CBC):** This tests for anemia, inflammation, and immune system problems.
- **Fasting vitamin B6:** This is a common and easily correctable nutrient deficiency. Levels above 30 nmol/l indicate adequate B6 status.
- **Homocysteine:** This tests for problems in pathways related to vitamin, amino acid, neurotransmitter, and antioxidant metabolism. Many things can cause a high homocysteine level, so if your level is too high (above 15 µmol/l), please consult with your health care practitioner.
- **Free carnitine:** If your carnitine level is low, you will have trouble burning fat for energy.
- **Celiac screening panel (if not done in the past three years):** This includes tissue transglutaminase IgA (anti-tTg) and total serum IgA tests.
- **Medication levels:** If you are taking any of the medications below, please have the level checked before you change your diet, especially if you move beyond the paleo diet to a low-carbohydrate, ketogenic, or carnivore diet.
 - lithium
 - clozapine
 - tricyclic antidepressants
 - anticonvulsant mood stabilizers (valproate, lamotrigine, etc.)⁵

Additional tests may be helpful depending on your personal health circumstances. Consult your health care professional.

Mental Health Self-Assessment Questionnaires

I recommend filling these out shortly before you begin your new diet and then again six to twelve weeks later. Assessment tools like these leave much to be desired, so if a question seems strange or doesn't seem to apply to you, you can either skip it or write in how you feel. After the first time you use them, you can ignore the time frames suggested on the tops of the questionnaires and simply use the previous week as your time frame instead.

- Modified Yale Food Addiction Scale-2.0 (MYFAS-2.0)
<https://sites.lsa.umich.edu/fastlab/yale-food-addiction-scale/>
- Altman Self-Rating Mania Scale (ASRM) <https://psychology-tools.com/test/altman-self-rating-mania-scale>
- Adult ADHD Self-Report Scale (ASRS)
<https://www.apaservices.org/practice/reimbursement/health-registry/self-reporting-symptom-scale.pdf>
- Beck Depression Inventory (BDI)
<https://www.ismanet.org/doctoryourspirit/pdfs/Beck-Depression-Inventory-BDI.pdf>
- Eating Disorder Diagnostic Scale (EDDS)
<http://www.ori.org/files/Static%20Page%20Files/EDDS.pdf>
- Generalized Anxiety Disorder Assessment-7 (GAD-7)
https://adaa.org/sites/default/files/GAD-7_Anxiety-updated_0.pdf
- Obsessive-Compulsive Inventory-Revised (OCI-R)
<https://simpleandpractical.com/wp-content/uploads/2020/02/OCI-R.pdf>
- Self-Administered Gerocognitive Exam (SAGE) test for cognitive impairment <https://wexnermedical.osu.edu/brain-spine-neuro/memory-disorders/sage/download-the-sage-test>

Footnote

i Note: fasting means nothing by mouth except water and medicines for 12–14 hours prior to the blood draw—no caffeine, no diet beverages, etc.

Appendix B

Selected Resources

You can find articles about nutrition science, ketogenic diets, and mental health on my website Diagnosis:Diet (<https://www.diagnosisdiet.com>).

General Nutrition Science and Metabolic Health

Big Fat Surprise by Nina Teicholz (Simon & Schuster, 2014)
Why We Get Sick by Benjamin Bikman (BenBella Books, 2020)
Good Calories, Bad Calories by Gray Taubes (Knopf, 2007)
Real Food for Pregnancy by Lily Nichols (Lily Nichols, 2018)
The Obesity Code by Jason Fung (Greystone, 2016)

Mental Health

Brain Energy by Christopher M. Palmer (BenBella, 2022)
The Alzheimer's Antidote by Amy Berger (Chelsea Green, 2017)
Grain Brain by David Perlmutter (Rev. ed. Little Brown Spark, 2018)
The End of Alzheimer's by Dale E. Bredesen (Avery, 2017)
Answers to Anorexia by James M. Greenblatt (2nd ed. Friesen Press, 2021)

Paleo Diets

The Paleo Diet by Loren Cordain (Wiley, 2011)
It Starts with Food by Hartwig Dallas and Melissa Urban (Victory Belt, 2014)
The Paleo Solution by Robb Wolf (Victory Belt, 2017)

COOKBOOKS

The Whole 30 by Melissa Hartwig Urban and Dallas Hartwig (Houghton

Mifflin Harcourt, 2015)
Nom Nom Paleo by Michelle Tam and Henry Fong (Andrews McMeel Publishing, 2013)

Ketogenic Diets

Metabolic Mind (<https://www.metabolicmind.org>): A pioneering non-profit initiative launched by the Baszucki Group to share information and resources about the emerging field of metabolic psychiatry.

The Virta Health blog (<https://www.virtahealth.com/blog>): Educational content produced by leading scientists in the field of metabolic health.

The Charlie Foundation for Ketogenic Therapies (<https://charliefoundation.org>): A veteran nonprofit organization with a focus on epilepsy care.

KetoMojo (<http://www.keto-mojo.com>): A content-rich website focused on ketogenic diets and ketone monitoring.

The Levels Health mental health blog (www.levelshealth.com/blog/category/mental-health)

Ken Berry MD's YouTube channel (<https://www.youtube.com/@KenDBerryMD>)

Ketogenic Diet and Metabolic Therapies by Susan A. Masino, ed. (2nd ed., Oxford, 2022)

Ketogenic Diet Therapies for Epilepsy and Other Conditions by Eric Kossoff MD, et al. (7th ed. Demos Health/Springer, 2021)

Ketogenic edited by Timothy Noakes and Nutrition Network (Academic Press, 2023)

The Case for Keto by Gary Taubes (Knopf, 2020)

COOKBOOKS

Maria Emmerich's collection of ketogenic cookbooks, especially *Easy Dairy-Free Ketogenic Recipes* (Victory Belt, 2018)

The Ketogenic Kitchen by Domini Kemp and Patricia Daly (Chelsea Green, 2016)

ATHLETIC PERFORMANCE

The Art and Science of Low Carbohydrate Performance by Jeff Volek and Stephen Phinney (Beyond Obesity, 2012)

CONTINUING EDUCATION RESOURCES FOR HEALTH PROFESSIONALS

Ketogenic Diets for Mental Health clinician training program

(<https://www.diagnosisdiet.com/training>) Taught by Dr. Georgia Ede; accredited for medical and nutrition professionals

Ketogenic Therapeutics Mastery (<https://www.ketomastery.pro>) Taught by Beth Zupec-Kania, RD and Denise Potter, RDN; accredited by the Academy of Nutrition and Dietetics

The European Keto-Live Centre (<https://www.european-keto-live-centre.com>) hosts live and recorded content focused on ketogenic metabolic therapies for noncommunicable diseases.

The Metabolic Health Initiative (<https://www.metabolicinitiative.com>) hosts live and digital content focused on nutrition, metabolism, clinical care, and human performance.

The Nutrition Network (nutrition-network.org) offers online trainings in low-carbohydrate nutrition for clinicians and coaches.

The Society of Metabolic Health Practitioners (<https://thesmhp.org>) offers clinical consensus guidelines, professional resources, educational content, and a path to accreditation for low-carbohydrate practitioners.

Treating Metabolic Syndrome, Type 2 Diabetes, and Obesity with Therapeutic Carbohydrate Restriction (<https://www.dietdoctor.com/cme>) is a free CME-course produced by Dr. Andreas Eenfeldt, Dr. Bret Scher, Dr. Adele Hite and Franziska Spritzler, RD, CDE.

CLINICIAN DIRECTORIES

The Ketogenic Diets for Mental Health Clinician Directory

(<https://www.diagnosisdiet.com/directory>), created and hosted by Dr. Ede; limited to practitioners who use ketogenic diets to treat mental health conditions.

Diet Doctor's low-carb clinician directory:

(<https://www.dietdoctor.com/low-carb/doctors>)

Society of Metabolic Health Practitioners low-carb clinician directory:

(<https://thesmhp.org/directory>)

MACRONUTRIENT CALCULATORS

Free online ketogenic diet calculators you can use to personalize your macronutrient ratios:

Maria Mind Body Health (<https://mariamindbodyhealth.com/new-keto-calculator>)

Keto-Mojo (<https://keto-mojo.com/mymojomacros-keto-macro-calculator>)

Carnivore Diets

Amber O’Hearn’s website (<https://www.mostly-fat.com/eat-meat-not-too-little-mostly-fat>)

Justmeat.co (<https://www.justmeat.co>): a compilation of carnivore resources

The Carnivore Diet by Shawn Baker (Victory Belt, 2019)

The Carnivore Cookbook by Maria Emmerich and Craig Emmerich (Victory Belt, 2020)

Miscellaneous

FOOD ADDICTION

Fork in the Road by Jen Unwin (FTR Publishing, 2021)

Food Junkies by Vera Tarman (2nd ed. Dundurn, 2019)

Bitten Jonsson, RN, Leg.SSK offers certification trainings in Holistic Addiction Medicine focused on food and sugar addiction (<https://www.bittensaddiction.com/en/professional-training>).

HISTAMINE INTOLERANCE

“Freshness Counts: Histamine Intolerance”

(<https://www.diagnosisdiet.com/full-article/histamine-intolerance>)

“Histamine Intolerance: Understanding the Science”

(<https://www.diagnosisdiet.com/full-article/histamine-intolerance-science>)

CHOLESTEROL

Lipid researcher Dave Feldman's website (<https://cholesterolcode.com>) educates, empowers, and supports people interested in understanding cholesterol testing

David M. Diamond, Benjamin T. Bikman, and Paul Mason, "Statin Therapy Is Not Warranted for a Person with High LDL-Cholesterol on a Low-Carbohydrate Diet." *Current Opinion in Endocrinology, Diabetes, and Obesity* 29, no. 5 (2022): 497–511,
<https://doi.org/10.1097/MED.0000000000000764>.

ETHICAL AND ENVIRONMENTAL CONCERNS ABOUT MEAT

Defending Beef by Nicolette Hahn Niman (Rev. ed. Chelsea Green, 2021)
Sacred Cow by Diana Rodgers and Robb Wolf (BenBella Books, 2020)
The Vegetarian Myth by Lierre Keith (Flashpoint Press, 2009)

BMI CALCULATOR

CDC's Adult BMI Calculator:

https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/bmi_calculator.html

Appendix C

Essential Micronutrients and Brain Metabolism

As discussed in [chapters 4](#) and [5](#), micronutrients are indispensable to the building and burning pathways that make up the miracle of brain metabolism. My goal in creating this resource was to help bring this generic list of players to life by describing the fascinating tasks each nutrient performs, paying special attention to the contributions each one makes to maintaining mental health.

Try not to think of this as a list of supplements—these are essential ingredients we are supposed to be able to obtain from our diets. We are best adapted to absorbing, utilizing, and achieving proper balance of these nutrients when we consume them in whole foods, not as isolated, concentrated extracts.

Vitamin A (retinol and related compounds): True vitamin A compounds (not to be confused with beta-carotene and similar compounds found in carrots and certain other colorful plant foods) belong to a family of fat-soluble hormones best known for their role in eye health, specifically for making both night vision and color perception possible.¹ Less commonly known is that vitamin A influences the genes involved in the growth and development of all cells, and therefore is crucial to the development and maintenance of the entire brain. Learning and memory also rely heavily on vitamin A to help connect neurons in new patterns to solidify knowledge.² Vitamin A deficiency in early life increases the risk for autism, and problems with vitamin A signaling may play a role in the development of schizophrenia. Vitamin A signaling can decline with age, contributing to cognitive deficits later in life.

The Busy B's: Even though they look nothing alike and have unique responsibilities, the B vitamins are often lumped together and referred to as “B complex,” because they all serve as coenzymes (enzyme assistants) that

help cells extract energy from food and assemble vital molecules, so they are indispensable to multiple burning and building pathways.³ We must consume foods rich in B vitamins regularly because most of them can't be stored in our tissues, with the notable exception of vitamin B12, which we store in the liver. Since the brain is a high-energy organ, even brief deficiency of a single member of the B vitamin family can slow the brain's machinery and lead to vague, nonspecific psychiatric symptoms, such as fatigue, apathy, or insomnia.

Vitamin B1 (thiamine) helps make and break the bonds that hold molecules together. It plays essential roles in both glycolysis (Engine G) and the citric acid cycle (part of Engine M), and connects the two pathways by converting pyruvate to acetyl CoA. It also helps the pentose phosphate pathway to make DNA and RNA, and assists in the construction of the neurotransmitters acetylcholine, glutamate, and GABA. Since glucose processing requires thiamine, diets high in carbohydrate increase thiamine requirements. Deficiency remains widespread in parts of the world that rely heavily on starchy staples like rice and have poor access to thiamine-rich protein sources.⁴

Vitamin B2 (riboflavin) is an integral part of FAD, which carries electrons to the electron transport chain. Riboflavin helps build antioxidants and synthesize vitamin B3, and is also required to activate vitamins B6 and B9.⁵

Vitamin B3 (niacin) is an integral part of NAD, which is used by hundreds of enzymes to help transfer electrons between molecules. Severe, advanced niacin deficiency causes pellagra, a disease which can lead to depression, psychosis, delirium, and dementia.

Vitamin B5 (pantothenic acid) is an integral part of CoA (coenzyme A), which changes the shape of large molecules to help them undergo chemical reactions more easily. Vitamin B5 participates widely in metabolism but is particularly important to the assembly of components needed for growth and is required to activate folate (vitamin B9).⁶

Vitamin B6 (pyridoxine) is used by dozens of enzymes to help transfer carbon building blocks between molecules. Vitamin B6 is required for gluconeogenesis, amino acid processing, and to make vitamin B3, DNA, RNA, serotonin, dopamine, norepinephrine, and GABA.⁷

Vitamin B7 (biotin) helps add carbon building blocks to molecules,

regulates gene activity, and supports gluconeogenesis, the production of blood glucose.⁸

Vitamin B9 (folate) supplies the carbon building blocks needed to help vitamin B12 make DNA, myelin, and certain neurotransmitters including serotonin, dopamine, and norepinephrine.⁹ Cells can't multiply without DNA, so folate requirements are much higher in times of rapid growth and development (such as pregnancy), and in parts of the body that replenish cells frequently, such as the bone marrow where red blood cells are produced. This is why folate deficiency can cause anemia (low red blood cell count) and neural tube defects such as spina bifida. Flour and cereal fortification programs in many countries have helped prevent deficiencies, but these use synthetic folic acid rather than the natural folate found in foods. Best animal food source is liver; good plant sources include spinach, asparagus, and avocado.

Vitamin B12 (cobalamin): Whereas most B vitamins participate in dozens to hundreds of metabolic reactions, B12 serves only two enzymes, so we need very little B12 and it can take years to deplete our reserves. The first enzyme helps vitamin B9 (folate) move carbon building blocks between molecules to make neurotransmitters and DNA (which is why B12 deficiency, like folate deficiency, can cause anemia), and the other enzyme is used to build myelin.¹⁰ Vitamin B12 deficiency is not uncommon even in affluent countries, partly because so many medications and health conditions can interfere with B12 absorption, and partly because diets low in animal foods are becoming more prevalent. B12 deficiency can lead to a variety of psychiatric symptoms, including depression, psychosis, memory problems, and personality changes.¹¹ B12 is only found in animal foods; good sources include shellfish, fish, and red meat.

Vitamin C (L-ascorbic acid) is a coenzyme required to build collagen (a component of the blood-brain barrier), and helps regulate the production of myelin which insulates brain circuits.¹²

Vitamin D3 (cholecalciferol): Strictly speaking, vitamin D3 is not essential in the diet because your skin can produce it if exposed to enough sunlight. Vitamin D is a fat-soluble hormone that influences brain development, calcium balance, antioxidant defenses, and neuroplasticity—the creation of new neuron networks in response to new experiences, which is key to learning and memory.¹³ Vitamin D deficiency is very common,

particularly in people with insulin resistance, and deficiency during pregnancy increases risk for autism.¹⁴ Good dietary sources include fish and pork.

Vitamin E (alpha-tocopherol) helps protect unsaturated fatty acids (MUFAs and PUFAs) from oxidative damage, helps maintain the shape of cell membranes, and regulates genes involved in protecting cell membrane function.¹⁵

Vitamin K1 helps add carbon groups to clotting proteins, allowing them to bind calcium and initiate the “coagulation cascade” to prevent uncontrolled bleeding.¹⁶

Vitamin K2 is a much-overlooked fat-soluble hormone that activates proteins involved in brain cell growth and survival and participates in the production of vital membrane fats (sphingolipids), including those needed to make myelin.¹⁷ Several different forms of K2 exist, but 98 percent of the K2 in the human brain exists in a form called MK-4.¹⁸ Best dietary sources are liver and egg yolks.

Calcium: Like a scout, calcium carries high-priority messages about energy demands, neurotransmitter supply, and cell health from the outer reaches of the neuron to deep inside the mitochondria and nucleus (the cell’s command center) so they can rapidly adapt to changing circumstances.¹⁹ Glutamate and GABA receptors use calcium, and calcium signaling is critical for learning and memory (neuroplasticity), neurotransmitter release, and even cell survival. If the cell is in dire straits due to viral infection, lack of oxygen, or other serious threats, large amounts of calcium will rush in, initiating the cell’s suicide program (apoptosis).²⁰

Chloride is the dominant negatively charged ion in the brain. It helps regulate fluid balance and cell volumes and cooperates with sodium to maintain neurons’ readiness to fire.²¹

Choline: The vast majority of choline is used to make phosphatidylcholine, an essential component of cell membranes. Choline is also used to build myelin, DNA molecules, and the neurotransmitter acetylcholine. Choline was only recognized as an essential nutrient in 1998, so little is known about how deficiency affects mental health, but early studies suggest that choline deficiency may affect attention and memory, perhaps because acetylcholine is so important to these brain functions.²²

Studies find that most people in the United States do not consume adequate choline; best sources are red meat, liver, eggs, and fish roe.

Copper: The electron transport chain relies on copper to pull electrons through one of its large enzyme complexes (cytochrome c oxidase) as it works to make ATP. The enzyme that transforms dopamine into norepinephrine depends on copper as well.²³

Iodine is an integral part of thyroid hormone, which is not only a major orchestrator of brain development in early life but also supports healthy brain metabolism throughout the life span.²⁴ Iodine deficiency causes hypothyroidism (low thyroid hormone activity); when this occurs during pregnancy, it can lead to irreversible cognitive deficits in the developing baby. Iodine deficiency is widespread, affecting up to two billion people, including in the United States and Europe, and is a leading cause of preventable intellectual disabilities worldwide.²⁵ In adults, hypothyroidism can cause symptoms of depression such as apathy and fatigue, and can even cause reversible dementia, likely due in part to sluggish brain glucose metabolism.²⁶ Best food sources are fish, shrimp, seaweed, and iodized salt.

Iron: When we think of iron, we think of blood, but this mineral's responsibilities extend far beyond carrying oxygen to the brain in red blood cells. Iron is gifted with the ability to exist in two different charged states, so it can give and receive electrons easily. This special talent makes it indispensable to the electron transport chain and many other pathways, including those used to construct DNA, myelin, and the neurotransmitters serotonin, dopamine, and norepinephrine.²⁷ Iron deficiency is the most common nutrient deficiency in the world, affecting more than 25 percent of the world's population, most of whom are pregnant women and very young children. Since iron is needed to build DNA and myelin, iron deficiency during pregnancy can have irreversible effects on a child's intelligence, memory, and attention, and can increase risk for autism and schizophrenia.²⁸ Best dietary sources are red meat, liver, mussels, and oysters.

Magnesium's compact size and strong positive charge give it magnetic properties useful in hundreds of chemical reactions, helping to generate energy, build proteins, and stabilize genes. Magnesium exists in balance with calcium and zinc, which keeps their destructive influences in check. One of magnesium's most intriguing tasks is to sit stubbornly inside

glutamate receptors (NMDA receptors, to be exact), plugging them up and preventing positive ions from entering the cell. Only when a strong electrical signal comes along will magnesium pop out like a champagne cork and allow those ions to pour in so the neuron can fire. NMDA receptors are particularly important for learning, memory, and healthy circadian rhythm (sleep-wake patterns).²⁹

Manganese: The antioxidant enzyme superoxide dismutase, which shields mitochondria from free radical damage, contains manganese. The multipurpose enzyme glutamine synthetase, which is used to manufacture glutamine, glutamate, and GABA, as well as to detoxify glutamate and ammonia in the brain,³⁰ also requires manganese.

Molybdenum: Only four enzymes in the body require molybdenum. These enzymes help prevent DNA mutations and support healthy uric acid levels (which protects the brain against oxidative stress).

Phosphorus is an essential component of cell membranes, DNA and RNA, ATP molecules (the P stands for phosphate), and bone. It also participates in multiple chemical reactions and helps regulate the pH of the blood.

Potassium is the dominant positively charged ion inside neurons, with concentrations maintained at roughly thirty times higher inside than outside to help maintain neurons' readiness to fire.³¹ The enzyme that releases energy from ATP also requires potassium.

Selenium: Several antioxidant enzymes contain selenium, including glutathione peroxidase, which helps protect the brain against stress, including oxidative stress.³²

Sodium is the dominant positively charged ion outside neurons, with concentrations maintained at roughly ten times higher outside than inside to help maintain neurons' readiness to fire.³³

Sulfur is an essential component of insulin and glutathione (one of the most important antioxidants in the brain). It is also required to build two amino acids (cysteine and methionine) and helps guide electrons through the electron transport chain.

Zinc allows certain proteins to fold into their correct shapes and assists certain enzymes in their catalytic duties. Zinc is required for healthy immune system function and neurotransmitter activity. One of zinc's unique responsibilities is to burst out of tiny storage compartments into the synapse

alongside glutamate (a stimulating neurotransmitter) to buffer its signal. Zinc also behaves as a natural dopamine reuptake inhibitor, prolonging dopamine signaling in the synapse. Zinc ripens young BDNF (brain derived growth factor) molecules to maturity so they may fertilize developing neurons, supporting the process of neuroplasticity. Zinc is also central to the process of autophagy; when mitochondria or other critical cell components are damaged beyond repair and need to be destroyed, zinc helps calcium flip the kill switch, partly by intentionally generating oxygen free radicals to attack them from within and finish them off.³⁴

Notes

Chapter 1

- [1](#) “Mental Disorder,” Fact Sheet, World Health Organization, last modified June 2, 2022, <https://www.who.int/news-room/fact-sheets/detail/mental-disorders>.
- [2](#) “Mental Health: Burden,” World Health Organization, published December 19, 2019, <https://www.who.int/health-topics/mental-health>.
- [3](#) “Mental Health: Burden.”
- [4](#) “The Deteriorating Mental Health of U.S. College Students: Part 1,” Imagine America, March 2, 2020, <https://www.imagine-america.org/deteriorating-mental-health-u-s-college-students-part/>.
- [5](#) Kelly Wester, Heather Trepal, and Kelly King, “Nonsuicidal Self-Injury: Increased Prevalence in Engagement,” *Suicide & Life-Threatening Behavior* 48, no. 6 (2018): 690–8, <https://doi.org/10.1111/sltb.12389>.
- [6](#) “450% Increase in Student Mental Health Declarations Over Last Decade but Progress Still Needed to Address Declarations Stigma,” Universities and Colleges Admissions Service, June 17, 2021, <https://www.ucas.com/corporate/news-and-key-documents/news/450-increase-student-mental-health-declarations-over-last-decade-progress-still-needed-address>.
- [7](#) Hui Zheng and Paola Echave, “Are Recent Cohorts Getting Worse? Trends in US Adult Physiological Status, Mental Health, and Health Behaviors Across a Century of Birth Cohorts,” *American Journal of Epidemiology* 190, no. 11 (2021): 2242–55, <https://doi.org/10.1093/aje/kwab076>.
- [8](#) A. M. Foerschner, “The History of Mental Illness: From *Skull Drills* to *Happy Pills*,” *Inquiries* 2, no. 9 (2010): 3–4, <http://www.inquiriesjournal.com/articles/1673/3/the-history-of-mental-illness-from-skull-drills-to-happy-pills>.
- [9](#) Henry Maudsley, *The Physiology and Pathology of the Mind* (New York: D. Appleton and Company, 1867), 201.
- [10](#) Anne Harrington, *Mind Fixers: Psychiatry’s Troubled Search for the Biology of Mental Illness* (New York: W. W. Norton & Company, 2019), chap. 1, EPUB.
- [11](#) Henrik Walter, “The Third Wave of Biological Psychiatry,” *Frontiers in*

- Psychology* 4 (2013): 582, <https://doi.org/10.3389/fpsyg.2013.00582>.
- 12 Edward Shorter, “The History of Lithium Therapy,” *Bipolar Disorders* 11, suppl 2 (2009): 4–9, <https://doi.org/10.1111/j.1399-5618.2009.00706.x>.
- 13 R. Cancro, “The Introduction of Neuroleptics: A Psychiatric Revolution,” *Psychiatric Services* 51, no. 3 (2000): 333–5, <https://doi.org/10.1176/appi.ps.51.3.333>.
- 14 Jerome Groopman, “The Troubled History of Psychiatry,” *The New Yorker*, May 20, 2019, <https://www.newyorker.com/magazine/2019/05/27/the-troubled-history-of-psychiatry>.
- 15 James McCormack and Christina Korownyk, “Effectiveness of Antidepressants,” *BMJ (Clinical Research edition)* 360 (2018): k1073, <https://doi.org/10.1136/bmj.k1073>.
- 16 McCormack and Korownyk, “Effectiveness of Antidepressants.”
- 17 Joanna Moncrieff et al., “The Serotonin Theory of Depression: A Systematic Umbrella Review of the Evidence,” *Molecular Psychiatry*, (2022), <https://doi.org/10.1038/s41380-022-01661-0>.
- 18 Stefan Leucht et al., “Sixty Years of Placebo-Controlled Antipsychotic Drug Trials in Acute Schizophrenia: Systematic Review, Bayesian Meta-Analysis, and Meta-Regression of Efficacy Predictors,” *American Journal of Psychiatry* 174, no. 10 (2017): 927–42, <https://doi.org/10.1176/appi.ajp.2017.16121358>.
- 19 Sung Woo Ahn et al., “Long-Term Response to Mood Stabilizer Treatment and Its Clinical Correlates in Patients with Bipolar Disorders: A Retrospective Observational Study,” *International Journal of Bipolar Disorders* 5, no. 1 (2017): 24, <https://doi.org/10.1186/s40345-017-0093-5>.
- 20 Roy H. Perlis et al., “Predictors of Recurrence in Bipolar Disorder: Primary Outcomes from the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD),” *American Journal of Psychiatry* 163 (2006): 217–24, <https://doi.org/10.1176/appi.ajp.163.2.217>.
- 21 Gregory A. Roth, “Global Burden of Cardiovascular Diseases and Risk Factors, 1990–2019: Update from the GBD 2019 Study,” *Cardiology* 76, no. 25 (2020): 2982–3021, <https://doi.org/10.1016/j.jacc.2020.11.010>.

- 22 Cheryl D. Fryar, Margaret D. Carroll, and Joseph Afful, “Prevalence of Overweight, Obesity, and Severe Obesity Among Adults Aged 20 and Over: United States, 1960–1962 Through 2017–2018,” *NCHS Health E-Stats*, 2020, <https://www.cdc.gov/nchs/data/hestat/obesity-adult-17-18/obesity-adult.htm>.
- 23 National Academies of Sciences, Engineering, and Medicine; Health and Medicine Division; Food and Nutrition Board; Roundtable on Obesity Solutions; Callahan EA, editor. *Current Status and Response to the Global Obesity Pandemic: Proceedings of a Workshop* (Washington (DC): National Academies Press, 2019) 2, Global Trends in Obesity, <https://www.ncbi.nlm.nih.gov/books/NBK544130>.
- 24 Cardiovascular disease: Sebastian Steven et al., “Vascular Inflammation and Oxidative Stress: Major Triggers for Cardiovascular Disease,” *Oxidative Medicine and Cellular Longevity* (2019): 7092151, <https://doi.org/10.1155/2019/7092151>; obesity: Prasenjit Manna and Sushil K. Jain, “Obesity, Oxidative Stress, Adipose Tissue Dysfunction, and the Associated Health Risks: Causes and Therapeutic Strategies,” *Metabolic Syndrome and Related Disorders* 13, no. 10 (2015): 423–44, <https://doi.org/10.1089/met.2015.0095>; type 2 diabetes: Antonio Ceriello and Enrico Motz, “Is Oxidative Stress the Pathogenic Mechanism Underlying Insulin Resistance, Diabetes, and Cardiovascular Disease? The Common Soil Hypothesis Revisited,” *Arteriosclerosis, Thrombosis, and Vascular Biology* 24, no. 5 (2004): 816–23, <https://doi.org/10.1161/01.ATV.0000122852.22604.78>.

Chapter 2

- [1](#) US Department of Agriculture and US Department of Health and Human Services, *Nutrition and Your Health: Dietary Guidelines for America* (Washington, D.C.: USDA, 1980), 1,
<https://www.dietaryguidelines.gov/sites/default/files/2019-05/1980%20DGA.pdf>.
- [2](#) David Grotto and Elisa Zied, “The Standard American Diet and Its Relationship to the Health Status of Americans,” *Nutrition in Clinical Practice* 25, no. 6 (2010): 603–12,
<https://doi.org/10.1177/0884533610386234>.
- [3](#) Grotto and Zied, “The Standard American Diet.”
- [4](#) Malcolm Peet, “Nutrition and Schizophrenia: An Epidemiological and Clinical Perspective,” *Nutrition and Health* 17, no. 3 (2003): 211–19,
<https://doi.org/10.1177/026010600301700304>.
- [5](#) Peet, “Nutrition and Schizophrenia.”
- [6](#) F. C. Dohan, et al., “Is Schizophrenia Rare If Grain Is Rare?” *Biological Psychiatry* 19, no. 3 (1984): 385–99.
- [7](#) Angelos K. Sikalidis, Anita H. Kelleher, and Aleksandra S. Kristo, “Mediterranean Diet,” *Encyclopedia* 1, no. 2 (2021): 371–87,
<https://doi.org/10.3390/encyclopedia1020031>.
- [8](#) Nina Teicholz, *The Big Fat Surprise* (New York: Simon & Schuster, 2014), 185–89.
- [9](#) Mauro Finicelli, Anna Di Salle, Umberto Galderisi, and Gianfranco Peluso, “The Mediterranean Diet: An Update of the Clinical Trials” *Nutrients* 14, no. 14 (2022): 2956, <https://doi.org/10.3390/nu14142956>.
- [10](#) Lisa Tussing-Humphreys et al., “Effect of Mediterranean Diet and Mediterranean Diet Plus Calorie Restriction on Cognition, Lifestyle, and Cardiometabolic Health: A Randomized Clinical Trial,” *Preventive Medicine Reports* 29 (2022): 101955,
<https://doi.org/10.1016/j.pmedr.2022.101955>.
- [11](#) Felice Jacka et al., “A Randomised Controlled Trial of Dietary Improvement for Adults with Major Depression (the ‘SMILES’ trial),” *BMC Medicine* 15, no. 1 (2017): 23, <https://doi.org/10.1186/s12916-017-0791-y>; Natalie Parletta et al., “A Mediterranean-Style Dietary

Intervention Supplemented with Fish Oil Improves Diet Quality and Mental Health in People with Depression: A Randomized Controlled Trial (HELPIMED)," *Nutritional Neuroscience* 22, no. 7 (2019): 474–87, <https://doi.org/10.1080/1028415X.2017.1411320>; Heather M. Francis et al., "A Brief Diet Intervention Can Reduce Symptoms of Depression in Young Adults—A Randomised Controlled Trial," *PloS ONE* 14, no. 10 (2019): e0222768, <https://doi.org/10.1371/journal.pone.0222768>.

- 12 Wolfgang Marx et al., "Diet and Depression: Exploring the Biological Mechanisms of Action," *Molecular Psychiatry* 26, no. 1 (2021): 134–50, <https://doi.org/10.1038/s41380-020-00925-x>.
- 13 Hadley Leggett and Shebani Sethi, "5 Questions: Shebani Sethi on the Connection between Metabolism and Mental Health," Stanford Medicine News Center, November 15, 2022, <https://med.stanford.edu/news/all-news/2022/11/metabolic-psychiatry.html>.
- 14 David Unwin et al., "What Predicts Drug-Free Type 2 Diabetes Remission? Insights from an 8-Year General Practice Service Evaluation of a Lower Carbohydrate Diet with Weight Loss," *BMJ Nutrition, Prevention & Health* 0 (2023): e000544, <https://doi.org/10.1136/bmjnph-2022-000544>.
- 15 Kirsty J. Martin-McGill et al., "Ketogenic Diets for Drug-Resistant Epilepsy," *The Cochrane Database of Systematic Reviews* 11, no. 11 (2018): CD001903, <https://doi.org/10.1002/14651858.CD001903.pub4>.
- 16 Tanya J. W. McDonald and Mackenzie C. Cervenka, "Ketogenic Diets for Adult Neurological Disorders," *Neurotherapeutics* 15, no. 4 (2018): 1018–31, <https://doi.org/10.1007/s13311-018-0666-8>; Diana Pietrzak et al., "The Therapeutic Role of Ketogenic Diet in Neurological Disorders," *Nutrients* 14, no. 9 (2022): 1952, <https://doi.org/10.3390/nu14091952>.

Chapter 3

- [1](#) Graham Sutton, “Putrid Gums and ‘Dead Men’s Cloaths’: James Lind aboard the *Salisbury*,” *Journal of the Royal Society of Medicine* 96, no. 12 (2003): 605–8, <https://doi.org/10.1177/014107680309601213>.
- [2](#) Kenneth J. Carpenter, *The History of Scurvy and Vitamin C* (Cambridge: Cambridge University Press, 1986), 253.
- [3](#) James A. Lind, *A Treatise of the Scurvy. In Three Parts. Containing an Inquiry into the Nature, Causes and Cure, of that Disease. Together with a Critical and Chronological View of What Has Been Published on the Subject* (Edinburgh: Sands, Murray and Cochran for A Kincaid and A Donaldson, 1753), 191.
- [4](#) Lind, *A Treatise of the Scurvy*, 192.
- [5](#) “Science and Technology,” *Oxford Reference*, accessed April 9, 2022, <https://www.oxfordreference.com/page/scienceandtech/science-and-technology>.
- [6](#) Kenneth J. Carpenter, “A Short History of Nutritional Science: Part 1 (1785–1885),” *The Journal of Nutrition* 133, no. 3 (March 2003): 638–45, <https://doi.org/10.1093/jn/133.3.638>.
- [7](#) Mohammad Hassan Murad, Shahnaz Sultan, Samir Haffar, and Fateh Bazerbachi, “Methodological Quality and Synthesis of Case Series and Case Reports,” *BMJ Evidence-Based Medicine* 23 (2018): 60–63, <http://dx.doi.org/10.1136/bmjebm-2017-110853>.
- [8](#) Matthew C. L. Phillips et al., “Randomized Crossover Trial of a Modified Ketogenic Diet in Alzheimer’s Disease,” *Alzheimer’s Research & Therapy* 13, no. 1 (2021): 51, <https://doi.org/10.1186/s13195-021-00783-x>.
- [9](#) Ambika Satija, Edward Yu, Walter C. Willett, and Frank B. Hu, “Understanding Nutritional Epidemiology and Its Role in Policy,” *Advances in Nutrition* 6, no. 1 (2015): 5–18, <https://doi.org/10.3945/an.114.007492>.
- [10](#) Daniel Steinberg, “In Celebration of the 100th Anniversary of the Lipid Hypothesis of Atherosclerosis,” *Journal of Lipid Research* 54, no. 11 (2013): 2946–9, <https://doi.org/10.1194/jlr.R043414>.
- [11](#) William E. Stehbens, “An Appraisal of Cholesterol Feeding in

- Experimental Atherogenesis," *Progress in Cardiovascular Diseases* 29, no. 2, (1986): 107–28, [https://doi.org/10.1016/0033-0620\(86\)90021-6](https://doi.org/10.1016/0033-0620(86)90021-6).
- 12 Aysha Akhtar, "The Flaws and Human Harms of Animal Experimentation," *Cambridge Quarterly of Healthcare Ethics* 24, no. 4 (2015): 407–19, <https://doi.org/10.1017/S0963180115000079>.
- 13 A. White and E. Ernst. "The Case for Uncontrolled Clinical Trials: A Starting Point for the Evidence Base for CAM," *Complementary Therapies in Medicine* 9, no. 2 (2001): 111–16, <https://doi.org/10.1054/ctim.2001.0441>.
- 14 Walter Willett, *Nutritional Epidemiology*, 3rd ed. (New York: Oxford University Press, 2013), 1.
- 15 Willett, *Nutritional Epidemiology*, 2.
- 16 Elizabeth E. Devore, Jae Hee Kang, Monique M. B. Breteler, and Francine Grodstein, "Dietary Intakes of Berries and Flavonoids in Relation to Cognitive Decline," *Annals of Neurology* 72, no. 1 (2012): 135–43, <https://doi.org/10.1002/ana.23594>.
- 17 Ryan Jaslow, "Eating Blueberries and Strawberries Staves Off Memory Decline, Study Suggests," *CBS News*, April 26, 2012, <https://www.cbsnews.com/news/eating-blueberries-and-strawberries-staves-off-memory-decline-study-suggests>.
- 18 Alice Park, "Brain Food: Berries Can Slow Cognitive Decline," *TIME*, April 26, 2012, <https://healthland.time.com/2012/04/26/brain-food-berries-can-slow-cognitive-decline>.
- 19 Jessica Maki, "Berries Keep Your Brain Sharp," *The Harvard Gazette*, April 26, 2012, <https://news.harvard.edu/gazette/story/2012/04/berries-keep-your-brain-sharp>.
- 20 H. Russell Bernard, Peter Killworth, David Kronenfeld, and Lee Sailer, "The Problem of Informant Accuracy: The Validity of Retrospective Data," *Annual Review of Anthropology* 13 (1984): 495–517, <https://doi.org/10.1146/annurev.an.13.100184.002431>.
- 21 David J. Mela and Jacqueline I. Aaron, "Honest But Invalid: What Subjects Say about Recording Their Food Intake," *Journal of the American Dietetic Association* 97, no. 7 (July 1997): 791–93.
- 22 Edgar Bright Wilson Jr., *An Introduction to Scientific Research* (New York: McGraw-Hill, 1952), 232.
- 23 *Merriam-Webster.com Dictionary*, s.v. "semiquantitative," accessed

August 29, 2023, <https://www.merriam-webster.com/dictionary/semiquantitative>.

- 24 Alessandra Malito, “Grocery Stores Carry 40,000 More Items Than They Did in the 1990s,” *MarketWatch*, June 17, 2017, <https://www.marketwatch.com/story/grocery-stores-carry-40000-more-items-than-they-did-in-the-1990s-2017-06-07>.
- 25 John P. Ioannidis, “The Challenge of Reforming Nutritional Epidemiologic Research,” *JAMA* 320, no. 10 (2018): 969–970, <https://doi.org/10.1001/jama.2018.11025>.
- 26 Satija, Yu, Willett, and Hu, “Understanding,” 8.
- 27 Austin Bradford Hill, “The Environment and Disease: Association or Causation?,” *Proceedings of the Royal Society of Medicine* 58 no. 5 (1965): 295–300.
- 28 Satija, Yu, Willett, and Hu, “Understanding,” 9.
- 29 Hill, “The Environment.”
- 30 Andrew Mente, Lawrence de Koning, Harry S. Shannon, and Sonia S. Anand, “A Systematic Review of the Evidence Supporting a Causal Link Between Dietary Factors and Coronary Heart Disease,” *Archives of Internal Medicine* 169, no. 7 (2009): 659–69, <https://doi.org/10.1001/archinternmed.2009.38>.
- 31 Ioannidis, “The Challenge,” E1.
- 32 Devore, Kang, Breteler, and Grodstein, “Dietary,” 135.
- 33 Park, “Brain Food.”
- 34 U.S. Department of Agriculture and U.S. Department of Health and Human Services, *Dietary Guidelines for Americans, 2020–2025*. 9th ed. (December 2020), <https://www.DietaryGuidelines.gov>.
- 35 IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, *Red Meat and Processed Meat*, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 114, (Lyon, France: International Agency for Research on Cancer, World Health Organization, 2018), <https://publications.iarc.fr/Book-And-Report-Series/Iarc-Monographs-On-The-Identification-Of-Carcinogenic-Hazards-To-Humans/Red-Meat-And-Processed-Meat-2018>. A summary article was published in 2015: Véronique Bouvard et al., “Carcinogenicity of Consumption of Red and Processed Meat,” *The Lancet. Oncology* 16, no. 16 (2015): 1599–600,

[https://doi.org/10.1016/S1470-2045\(15\)00444-1](https://doi.org/10.1016/S1470-2045(15)00444-1).

- 36 Walter Willett et al., “Food in the Anthropocene: The EAT-Lancet Commission on Healthy Diets from Sustainable Food Systems,” *Lancet (London, England)* 393, no. 10170 (2019): 447–92, [https://www.doi.org/10.1016/S0140-6736\(18\)31788-4](https://www.doi.org/10.1016/S0140-6736(18)31788-4).
- 37 American Society for Nutrition, “Millions of Cardiovascular Deaths Attributed to Not Eating Enough Fruits and Vegetables,” *American Society for Nutrition*, June 8, 2019, <https://nutrition.org/millions-of-cardiovascular-deaths-attributed-to-not-eating-enough-fruits-and-vegetables>.
- 38 Cheryl Bond-Nelms, “Your Fries May Be Deadly,” *AARP*, June 13, 2017, <https://www.aarp.org/health/healthy-living/info-2017/french-fries-bad-for-health-fd.html>.
- 39 University College London, “People Who Eat Dark Chocolate Less Likely to Be Depressed,” *ScienceDaily*, August 2, 2019, <https://www.sciencedaily.com/releases/2019/08/190802145458.htm>.

Chapter 4

- [1](#) Elizabeth M. Lillie et al., “Evaluation of Skull Cortical Thickness Changes with Age and Sex from Computed Tomography Scans,” *Journal of Bone and Mineral Research: The Official Journal of the American Society for Bone and Mineral Research* 31, no. 2 (2016): 299–307, <https://doi.org/10.1002/jbmr.2613>.
- [2](#) Steven T. Proulx, “Cerebrospinal Fluid Outflow: A Review of the Historical and Contemporary Evidence for Arachnoid Villi, Perineural Routes, and Dural Lymphatics,” *Cellular and Molecular Life Sciences: CMLS* 78, no. 6 (2021): 2429–457, <https://doi.org/10.1007/s00018-020-03706-5>.
- [3](#) Lauren N. Telano and Stephen Baker, “Physiology, Cerebral Spinal Fluid,” in *StatPearls* (Treasure Island, FL: StatPearls Publishing, Jan 2022–), updated July 9, 2021, <https://www.ncbi.nlm.nih.gov/books/NBK519007>.
- [4](#) Richard Daneman and Prat Alexandre, “The Blood-Brain Barrier,” *Cold Spring Harbor Perspectives in Biology* 7, no. 1 (2015): a020412, <https://doi.org/10.1101/cshperspect.a020412>.
- [5](#) Michel A. Hofman, “Evolution of the Human Brain: When Bigger Is Better,” *Frontiers in Neuroanatomy* 8 no. 15 (2014), <https://doi.org/10.3389/fnana.2014.00015>.
- [6](#) Telano and Baker, “Physiology.”
- [7](#) Jasleen Kaur et al., “Waste Clearance in the Brain,” *Frontiers in Neuroanatomy* 15, no. 665803 (2021), <https://doi.org/10.3389/fnana.2021.665803>.
- [8](#) Ruth O’Gorman Tuura et al., “Sleep-Related and Diurnal Effects on Brain Diffusivity and Cerebrospinal Fluid Flow,” *NeuroImage* 241 (2021): 118420, <https://doi.org/10.1016/j.neuroimage.2021.118420>.
- [9](#) Frederico A. C. Azevedo et al., “Equal Numbers of Neuronal and Nonneuronal Cells Make the Human Brain an Isometrically Scaled-Up Primate Brain,” *The Journal of Comparative Neurology* 513, no. 5 (2009): 532–541, <https://doi.org/10.1002/cne.21974>.
- [10](#) Christopher S. von Bartheld, Jami Bahney, and Suzana Herculano-Houzel, “The Search for True Numbers of Neurons and Glial Cells in

- the Human Brain: A Review of 150 Years of Cell Counting,” *Journal of Comparative Neurology*, 524, no. 18 (2016): 3865–3895, <https://doi.org/10.1002/cne.24040>.
- 11 Krishnagopal Dharani, “Chapter 2: Physiology of a Neuron,” in *The Biology of Thought: A Neuronal Mechanism in the Generation of Thought—A New Molecular Model* (Internet: Elsevier, 2014), <http://dx.doi.org/10.1016/B978-0-12-800900-0.00002-6>.
- 12 Krishnagopal Dharani, “Chapter 6: Dendrites and Primary Thought,” in *The Biology of Thought: A Neuronal Mechanism in the Generation of Thought—A New Molecular Model* (Internet: Elsevier, 2014), <http://dx.doi.org/10.1016/B978-0-12-800900-0.00006-3>.
- 13 Aliya L. Frederick and Gregg D. Stanwood, “Drugs, Biogenic Amine Targets and the Developing Brain,” *Developmental Neuroscience* 31, no. 1–2, (2009): 7–22, <https://doi.org/10.1159/000207490>.
- 14 C. Fernando Valenzuela, Michael P. Puglia, and Stefano Zucca, “Focus On: Neurotransmitter Systems,” *Alcohol Research & Health: the Journal of the National Institute on Alcohol Abuse and Alcoholism* 34, no. 1 (2011):106–120.
- 15 Frederick and Stanwood, “Drugs.”
- 16 Yann S. Mineur and Marina R Picciotto, “The Role of Acetylcholine in Negative Encoding Bias: Too Much of a Good Thing?,” *The European Journal of Neuroscience* 53, no. 1 (2021): 114–125, <https://doi.org/10.1111/ejn.14641>; Juhee Haam and Jerrel L. Yakel, “Cholinergic Modulation of the Hippocampal Region and Memory Function,” *Journal of Neurochemistry* 142, Suppl 2 (2017): 111–121, <https://doi.org/10.1111/jnc.14052>.
- 17 Sheng Peng et al., “Glutamate Receptors and Signal Transduction in Learning and Memory,” *Molecular Biology Reports* 38, no. 1 (2010): 453–460, <https://doi.org/10.1007/s11033-010-0128-9>.
- 18 Kresimir Krnjević, “How Does a Little Acronym Become a Big Transmitter?” *Biochemical Pharmacology* 68, no. 8 (2004): 1549–55, <https://doi.org/10.1016/j.bcp.2004.06.038>.
- 19 Joanna Moncrieff et al., “The Serotonin Theory of Depression: A Systematic Umbrella Review of the Evidence,” *Molecular Psychiatry*, (2022), <https://doi.org/10.1038/s41380-022-01661-0>.
- 20 Bo Wang et al., “Firing Frequency Maxima of Fast-Spiking Neurons in

- Human, Monkey, and Mouse Neocortex,” *Frontiers in Cellular Neuroscience* 10 (2016): 239, <https://doi.org/10.3389/fncel.2016.00239>.
- 21 Mikael Simons and Klaus-Armin Nave, “Oligodendrocytes: Myelination and Axonal Support,” *Cold Spring Harbor Perspectives in Biology* 8, no. 1 (2015): a020479, <https://doi.org/10.1101/cshperspect.a020479>.
- 22 Celeste Silveira et al., “Neuropsychiatric Symptoms of Multiple Sclerosis: State of the Art,” *Psychiatry Investigation* 16, no. 12 (2019): 877–88, <https://doi.org/10.30773/pi.2019.0106>.
- 23 Yonghee Kim, Jinhong Park, Yoon Kyung Choi, “The Role of Astrocytes in the Central Nervous System Focused on BK Channel and Heme Oxygenase Metabolites: A Review,” *Antioxidants* 8, no. 5 (2019): 121, <https://doi.org/10.3390/antiox8050121>.
- 24 Debasis Nayak, Theodore L. Roth, and Dorian B. McGavern, “Microglia Development and Function,” *Annual Review of Immunology* 32 (2014): 367–402, <https://doi.org/10.1146/annurev-immunol-032713-120240>.
- 25 Elena A. Ponomarenko et al., “The Size of the Human Proteome: The Width and Depth,” *International Journal of Analytical Chemistry* 2016 (2016): 7436849, <https://doi.org/10.1155/2016/7436849>.
- 26 Harris Ripps and Wen Shen, “Review: Taurine: A “Very Essential” Amino Acid,” *Molecular Vision* 18 (2012): 2673–86.
- 27 Steven R. Hertzler, Jacqueline C. Lieblein-Boff, Mary Weiler, and Courtney Allgeier, “Plant Proteins: Assessing Their Nutritional Quality and Effects on Health and Physical Function,” *Nutrients* 12, no. 12 (2020): 3704, <https://doi.org/10.3390/nu12123704>.
- 28 H. McIlwain and H. S. Bachelard, *Biochemistry and the Central Nervous System* (Edinburgh: Churchill Livingstone, 1985).
- 29 Johannes Weickenmeier et al., “The Mechanical Importance of Myelination in the Central Nervous System,” *Journal of the Mechanical Behavior of Biomedical Materials* 76 (2017): 119–124, <https://doi.org/10.1016/j.jmbbm.2017.04.017>.
- 30 Prasanna Kandel et al., “Oleic Acid Is an Endogenous Ligand of TLX/NR2E1 That Triggers Hippocampal Neurogenesis,” *Proceedings of the National Academy of Sciences of the United States of America* 119, no. 13 (2022): e2023784119, <https://doi.org/10.1073/pnas.2023784119>.
- 31 Timothy J. Tracey, Frederik J. Steyn, Ernst J. Wolvetang, and Shyuan T. Ngo, “Neuronal Lipid Metabolism: Multiple Pathways Driving

- Functional Outcomes in Health and Disease,” *Frontiers in Molecular Neuroscience* 11 (2018): 10, <https://doi.org/10.3389/fnmol.2018.00010>.
- 32 Michael A. Crawford, Walter F. Schmidt, C. Leigh Broadhurst, and Yiqun Wang, “Lipids in the Origin of Intracellular Detail and Speciation in the Cambrian Epoch and the Significance of the Last Double Bond of Docosahexaenoic Acid in Cell Signaling,” *Prostaglandins, Leukotrienes, and Essential Fatty Acids* 166 (2021): 102230, <https://doi.org/10.1016/j.plefa.2020.102230>.
- 33 Crawford, Schmidt, Broadhurst, and Wang, “Lipids,” 1.
- 34 Akhlaq A. Farooqui, Lloyd A. Horrocks, and Tahira Farooqui, “Modulation of Inflammation in Brain: A Matter of Fat,” *Journal of Neurochemistry* 101, no. 3 (2007): 577–99, <https://doi.org/10.1111/j.1471-4159.2006.04371.x>.
- 35 Wade T. Johnson et al., “Lipid-Based Regulators of Immunity,” *Bioengineering & Translational Medicine* 7, no. 2 (2021):e10288, <https://doi.org/10.1002/btm2.10288>.
- 36 Ann G. Liu et al., “A Healthy Approach to Dietary Fats: Understanding the Science and Taking Action to Reduce Consumer Confusion,” *Nutrition Journal* 16, no. 1 (2017): 53, <https://doi.org/10.1186/s12937-017-0271-4>.
- 37 Uram Jin, Soo Jin Park, and Sang Myun Park, “Cholesterol Metabolism in the Brain and Its Association with Parkinson’s Disease,” *Experimental Neurobiology* 28, no. 5 (2019): 554–67, <https://doi.org/10.5607/en.2019.28.5.554>.
- 38 Jin, Park, and Park, “Cholesterol Metabolism in the Brain.”

Chapter 5

- [1](#) Zhuo Fu, Elizabeth R. Gilbert, and Dongmin Liu, “Regulation of Insulin Synthesis and Secretion and Pancreatic Beta-Cell Dysfunction in Diabetes,” *Current Diabetes Reviews* 9, no. 11 (2014): 25–53.
- [2](#) Tiannan Wang et al., “Current Understanding of Glucose Transporter 4 Expression and Functional Mechanisms,” *World Journal of Biological Chemistry* 11, no. 3 (2020): 76–98,
<https://doi.org/10.4331/wjbc.v11.i3.76>.
- [3](#) Sander Kersten, “The Impact of Fasting on Adipose Tissue Metabolism,” *Biochimica et biophysica acta. Molecular and Cell Biology of Lipids* 1868, no. 3 (2023): 159262,
<https://doi.org/10.1016/j.bbalip.2022.159262>.
- [4](#) Jørgen Jensen, Per Inge Rustad, Anders Jensen Kolnes, and Yu-Chiang Lai, “The Role of Skeletal Muscle Glycogen Breakdown for Regulation of Insulin Sensitivity by Exercise.” *Frontiers in Physiology* 2, no. 112 (2011), <https://doi.org/10.3389/fphys.2011.00112>.
- [5](#) Ann G. Liu et al. “A Healthy Approach to Dietary Fats: Understanding the Science and Taking Action to Reduce Consumer Confusion,” *Nutrition Journal* 16, no. 1 (2017): 53, <https://doi.org/10.1186/s12937-017-0271-4>.
- [6](#) Laura R. Rich, William Harris, and Angus M. Brown, “The Role of Brain Glycogen in Supporting Physiological Function,” *Frontiers in Neuroscience* 13 (2019): 1176,
<https://doi.org/10.3389/fnins.2019.01176>.
- [7](#) Peter Schönfeld and Georg Reiser, “Brain Energy Metabolism Spurns Fatty Acids as Fuel Due to Their Inherent Mitotoxicity and Potential Capacity to Unleash Neurodegeneration,” *Neurochemistry International* 109 (2017): 68–77, <https://doi.org/10.1016/j.neuint.2017.03.018>.
- [8](#) I. Fritzsche, P. Bührdel, R. Melcher, and H. J. Böhme, “Stability of Ketone Bodies in Serum in Dependence on Storage Time and Storage Temperature,” *Clinical Laboratory* 47, no. 7–8 (2001): 399–403.
- [9](#) Alexandre Courchesne-Loyer et al., “Inverse Relationship between Brain Glucose and Ketone Metabolism in Adults during Short-Term Moderate Dietary Ketosis: A Dual Tracer Quantitative Positron

- Emission Tomography Study," *Journal of Cerebral Blood Flow and Metabolism* 37, no. 7 (2017): 2485–93, <https://doi.org/10.1177/0271678X16669366>.
- 10 Janice J. Hwang et al., "Blunted Rise in Brain Glucose Levels during Hyperglycemia in Adults with Obesity and T2DM," *JCI Insight* 2, no. 20 (2017): e95913, <https://doi.org/10.1172/jci.insight.95913>.
- 11 Shayne Mason, "Lactate Shuttles in Neuroenergetics-Homeostasis, Allostasis and Beyond," *Frontiers in Neuroscience* 11 (2017): 43, <https://doi.org/10.3389/fnins.2017.00043>.
- 12 Hermann Koepsell, "Glucose Transporters in Brain in Health and Disease," *Pflugers Archiv: European Journal of Physiology* 472, no. 9 (2020): 1299–343, <https://doi.org/10.1007/s00424-020-02441-x>.
- 13 Stephen C. Cunnane et al., "Can Ketones Compensate for Deteriorating Brain Glucose Uptake During Aging? Implications for the Risk and Treatment of Alzheimer's Disease." *Annals of the New York Academy of Sciences* 1367, no. 1 (2016): 12–20, <https://doi.org/10.1111/nyas.12999>.
- 14 Alexandre Courchesne-Loyer et al., "Inverse Relationship."
- 15 Mark P. Mattson et al., "Intermittent Metabolic Switching, Neuroplasticity and Brain Health," *Nature Reviews: Neuroscience* 19, no. 2 (2018): 63–80, <https://doi.org/10.1038/nrn.2017.156>.
- 16 Xiao-Hong Zhu et al., "Quantitative Imaging of Energy Expenditure in Human Brain," *NeuroImage* 60, no. 4 (2012): 2107–17, <https://doi.org/10.1016/j.neuroimage.2012.02.013>.
- 17 Miroslav Oborník, "Organellar Evolution: A Path from Benefit to Dependence," *Microorganisms* 10, no. 1 (2022): 122, <https://doi.org/10.3390/microorganisms10010122>.
- 18 Oborník, "Organellar Evolution."
- 19 Shona A. Mookerjee, Akos A. Gerencser, David G. Nicholls, and Martin D. Brand, "Quantifying Intracellular Rates of Glycolytic and Oxidative ATP Production and Consumption Using Extracellular Flux Measurements." *The Journal of Biological Chemistry* 292, no. 17 (2017): 7189–207, <https://doi.org/10.1074/jbc.M116.774471>.
- 20 Thomas Misgeld and Thomas L. Schwarz, "Mitostasis in Neurons: Maintaining Mitochondria in an Extended Cellular Architecture," *Neuron* 96, no. 3 (2017): 651–66, <https://doi.org/10.1016/j.neuron.2017.09.055>.

- 21 Vincent J. Miller, Frederick A. Villamena, and Jeff S. Volek, “Nutritional Ketosis and Mitohormesis: Potential Implications for Mitochondrial Function and Human Health.” *Journal of Nutrition and Metabolism* 2018 (2018): 5157645, <https://doi.org/10.1155/2018/5157645>.
- 22 Yuri Zilberman and Tanya Zilberman, “Glucose-Sparing Action of Ketones Boosts Functions Exclusive to Glucose in the Brain,” *eNeuro* 7, no. 6 (2020), <https://doi.org/10.1523/ENEURO.0303-20.2020>.
- 23 S. J. Kierans and C. T. Taylor, “Regulation of Glycolysis by the Hypoxia-Inducible Factor (HIF): Implications for Cellular Physiology,” *The Journal of Physiology* 599, no. 1 (2021): 23–37, <https://doi.org/10.1113/JP280572>.
- 24 Eloïse de Tredern et al., “Glial Glucose Fuels the Neuronal Pentose Phosphate Pathway for Long-Term Memory,” *Cell Reports* 36, no. 8 (2021): 109620, <https://doi.org/10.1016/j.celrep.2021.109620>.
- 25 Paul Trayhurn, “Oxygen-A Critical, but Overlooked, Nutrient,” *Frontiers in Nutrition* 6 (2019): 10, <https://doi.org/10.3389/fnut.2019.00010>.
- 26 Samina Salim, “Oxidative Stress and the Central Nervous System,” *The Journal of Pharmacology and Experimental Therapeutics* 360, no. 1 (2017): 201–5, <https://doi.org/10.1124/jpet.116.237503>.
- 27 Richard L. Veech, “The Therapeutic Implications of Ketone Bodies: The Effects of Ketone Bodies in Pathological Conditions: Ketosis, Ketogenic Diet, Redox States, Insulin Resistance, and Mitochondrial Metabolism,” *Prostaglandins, Leukotrienes, and Essential Fatty Acids* 70, no. 3 (2004): 309–19, <https://doi.org/10.1016/j.plefa.2003.09.007>.

Chapter 6

- [1](#) Irit Zohar et al., “Evidence for the Cooking of Fish 780,000 Years Ago at Gesher Benot Ya’aqov, Israel,” *Nature Ecology & Evolution* 6, (2022): 2016–28, <https://doi.org/10.1038/s41559-022-01910-z>.
- [2](#) Amaia Arranz-Otaegui et al., “Archaeobotanical Evidence Reveals the Origins of Bread 14,400 Years Ago in Northeastern Jordan,” *Proceedings of the National Academy of Sciences of the United States of America* 115, no. 31 (2018): 7925–30, <https://doi.org/10.1073/pnas.1801071115>.
- [3](#) Sarah B. McClure et al., “Fatty Acid Specific $\delta^{13}\text{C}$ Values Reveal Earliest Mediterranean Cheese Production 7,200 Years Ago,” *PLoS ONE* 13, no. 9 (2018): e020280, <https://doi.org/10.1371/journal.pone.0202807>.
- [4](#) Patricia Huebbe and Gerald Rimbach, “Historical Reflection of Food Processing and the Role of Legumes as Part of a Healthy Balanced Diet,” *Foods* 9, no. 8 (2020): 1056, <https://doi.org/10.3390/foods9081056>.
- [5](#) Carlos Augusto Monteiro et al., *Ultra-Processed Foods, Diet Quality, and Health Using the NOVA Classification System*. (Rome: FAO, 2019), 8, <https://www.fao.org/3/ca5644en/ca5644en.pdf>.
- [6](#) “Apparent Consumption of Selected Foodstuffs, Australia,” Australian Bureau of Statistics, March 30, 2022, <https://www.abs.gov.au/statistics/health/health-conditions-and-risks/apparent-consumption-selected-foodstuffs-australia/latest-release#summary>.
- [7](#) Carlos Augusto Monteiro et al., “Household Availability of Ultra-Processed Foods and Obesity in Nineteen European Countries,” *Public Health Nutrition* 21, no. 1 (2018): 18–26, <https://doi.org/10.1017/S1368980017001379>.
- [8](#) Federation of American Societies for Experimental Biology, “Highly Processed Foods Dominate U.S. Grocery Purchases,” ScienceDaily, March 29, 2015, www.sciencedaily.com/releases/2015/03/150329141017.htm.
- [9](#) Khalil Gibran Muhammad, “The Barbaric History of Sugar in America,”

The New York Times, August 14, 2019,
<https://www.nytimes.com/interactive/2019/08/14/magazine/sugar-slave-trade-slavery.html>.

- 10 Gary Taubes, *A Case Against Sugar* (New York: Alfred K. Knopf, 2016), 57.
- 11 Robert H. Lustig, *Metabolical: The Lure and the Lies of Processed Food, Nutrition, and Modern Medicine* (New York: Harper Wave, 2021), 221.
- 12 “What Are Whole Grains,” Ask USDA, November 4, 2022, <https://ask.usda.gov/s/article/What-are-whole-grains>.
- 13 Lisa M. Sanders et al., “Whole Grain Intake, Compared to Refined Grain, Improves Postprandial Glycemia and Insulinemia: A Systematic Review and Meta-Analysis of Randomized Controlled Trials,” *Critical Reviews in Food Science and Nutrition*, (2021): 1–19, <https://doi.org/10.1080/10408398.2021.2017838>; Kathy Musa-Veloso et al., “A Systematic Review and Meta-Analysis of Randomized Controlled Trials on the Effects of Oats and Oat Processing on Postprandial Blood Glucose and Insulin Responses,” *The Journal of Nutrition* 151, no. 2 (2021): 341–51, <https://doi.org/10.1093/jn/nxaa349>.
- 14 Eugenio Butelli et al., “Noemi Controls Production of Flavonoid Pigments and Fruit Acidity and Illustrates the Domestication Routes of Modern Citrus Varieties,” *Current Biology* 29, no. 1 (2019): 158–164.e2, <https://doi.org/10.1016/j.cub.2018.11.040>.
- 15 Josh Sosland, “US Sees Gain in Per Capita Flour Consumption,” Whole-Grain.com, May 18, 2023, <https://www.world-grain.com/articles/18528-us-sees-gain-in-per-capita-flour-consumption>.
- 16 Lisa M. Sanders et al., “Effects of Whole Grain Intake, Compared with Refined Grain, on Appetite and Energy Intake: A Systematic Review and Meta-Analysis,” *Advances in Nutrition* 12, no. 4 (2021): 1177–95, <https://doi.org/10.1093/advances/nmaa178>.
- 17 Charles Watt, Elizabeth Sanchez-Rangel, and Janice Jin Hwang, “Glycemic Variability and CNS Inflammation: Reviewing the Connection,” *Nutrients* 12, no. 12 (2020): 3906. 21 Dec. 2020, <https://doi.org/10.3390/nu12123906>.
- 18 Yuriko Kikkawa et al., “The Acute Effects of Glycemic Control on Nerve Conduction in Human Diabetics,” *Clinical Neurophysiology* 116,

- no. 2 (2005), 270–4, <https://doi.org/10.1016/j.clinph.2004.08.011>.
- 19 Louis Monnier et al., “Activation of Oxidative Stress by Acute Glucose Fluctuations Compared with Sustained Chronic Hyperglycemia in Patients with Type 2 Diabetes,” *JAMA* 295, no. 14 (2006): 1681–7, <https://doi.org/10.1001/jama.295.14.1681>.
- 20 Salvatore Bongarzone, Vilius Savickas, Federico Luzi, and Antony D. Gee, “Targeting the Receptor for Advanced Glycation Endproducts (RAGE): A Medicinal Chemistry Perspective,” *Journal of Medicinal Chemistry* 60, no. 17 (2017): 7213–32, <https://doi.org/10.1021/acs.jmedchem.7b00058>.
- 21 Damon DiSabato, Ning Quan, and Jonathan P. Godbout, “Neuroinflammation: The Devil Is in the Details,” *Journal of Neurochemistry* 139, Suppl 2 (2016): 136–53, <https://doi.org/10.1111/jnc.13607>.
- 22 Souhel Najjar et al., “Neuroinflammation and Psychiatric Illness,” *Journal of Neuroinflammation* 10, no. 43 (2013), <https://doi.org/10.1186/1742-2094-10-43>.
- 23 Romain Troubat et al., “Neuroinflammation and Depression: A Review,” *The European Journal of Neuroscience* 53, no. 1 (2021): 151–71, <https://doi.org/10.1111/ejn.14720>.
- 24 Ole Köhler et al., “Effect of Anti-Inflammatory Treatment on Depression, Depressive Symptoms, and Adverse Effects: A Systematic Review and Meta-Analysis of Randomized Clinical Trials,” *JAMA Psychiatry* 71, no. 12 (2014): 1381–91, <https://doi.org/10.1001/jamapsychiatry.2014.1611>.
- 25 Köhler, “Effect of Anti-Inflammatory.”
- 26 Sang Won Jeon and Yong-Ku Kim, “Inflammation-Induced Depression: Its Pathophysiology and Therapeutic Implications,” *Journal of Neuroimmunology* 313 (2017): 92–8, <https://doi.org/10.1016/j.jneuroim.2017.10.016>.
- 27 O. Köhler-Forsberg et al., “Efficacy of Anti-Inflammatory Treatment on Major Depressive Disorder or Depressive Symptoms: Meta-Analysis of Clinical Trials,” *Acta Psychiatrica Scandinavica* 139, no. 5 (2019): 404–19, <https://doi.org/10.1111/acps.13016>.
- 28 Akito Nakao, Yoshihiro Matsunaga, Katsumi Hayashida, and Nobuaki Takahashi, “Role of Oxidative Stress and Ca²⁺ Signaling in Psychiatric

- Disorders," *Frontiers in Cell and Developmental Biology* 9 (2021): 615569, <https://doi.org/10.3389/fcell.2021.615569>.
- 29 Geon Ha Kim et al., "The Role of Oxidative Stress in Neurodegenerative Diseases," *Experimental Neurobiology* 24, no. 4 (2015): 325–40, <https://doi.org/10.5607/en.2015.24.4.325>.
- 30 Nina Teicholz, *Big Fat Surprise*, (New York: Simon & Schuster, 2014), 84.
- 31 Teicholz, *Big Fat Surprise*, 87.
- 32 Teicholz, *Big Fat Surprise*, 47–48.
- 33 Albert Dijkstra and G. van Duijn, "Vegetable Oils: Oil Production and Processing," in *Encyclopedia of Food and Health*, eds. Benjamin Caballero, Paul M. Finglas, and Fidel Toldrá (2016): 373–80, <https://doi.org/10.1016/B978-0-12-384947-2.00707-8>.
- 34 Simon C. Dyall et al., "Polyunsaturated Fatty Acids and Fatty Acid-Derived Lipid Mediators: Recent Advances in the Understanding of Their Biosynthesis, Structures, and Functions," *Progress in Lipid Research* 86 (2022): 101165, <https://doi.org/10.1016/j.plipres.2022.101165>.
- 35 Donghee Kim, Jeong-Eun Choi, and Yongsoon Park, "Low-Linoleic Acid Diet and Oestrogen Enhance the Conversion of α-Linolenic Acid into DHA through Modification of Conversion Enzymes and Transcription Factors," *British Journal of Nutrition* 121, no. 2 (2019): 137–45, <https://doi.org/10.1017/S0007114518003252>.
- 36 Abeba Haile Mariamenatu and Emebet Mohammed Abdu, "Overconsumption of Omega-6 Polyunsaturated Fatty Acids (PUFAs) Versus Deficiency of Omega-3 PUFAs in Modern-Day Diets: The Disturbing Factor for Their 'Balanced Antagonistic Metabolic Functions' in the Human Body," *Journal of Lipids* 2021 (2021): 8848161, <https://doi.org/10.1155/2021/8848161>.
- 37 Klaus W. Lange, "Omega-3 Fatty Acids and Mental Health," *Global Health Journal* 4, no. 1 (2020): 18–30, <https://doi.org/10.1016/j.glohj.2020.01.004>.
- 38 Christopher E. Ramsden et al., "Dietary Alteration of n-3 and n-6 Fatty Acids for Headache Reduction in Adults with Migraine: Randomized Controlled Trial," *BMJ (Clinical research)* 374 (2021): n1448, <https://doi.org/10.1136/bmj.n1448>.

- 39 Jakob S. Hamilton and Eric L. Klett. “Linoleic Acid and the Regulation of Glucose Homeostasis: A Review of the Evidence,” *Prostaglandins, Leukotrienes, and Essential Fatty Acids* 175 (2021): 102366, <https://doi.org/10.1016/j.plefa.2021.102366>.
- 40 Ameer Y. Taha, “Linoleic Acid—Good or Bad for the Brain?,” *NPJ Science of Food* 4 (2020): 1, <https://doi.org/10.1038/s41538-019-0061-9>
- 41 K. Allison Amick et al., “Plasma Glycocholic Acid and Linoleic Acid Identified as Potential Mediators of Mitochondrial Bioenergetics in Alzheimer’s Dementia,” *Frontiers in Aging Neuroscience* 14 (2022): 954090, <https://doi.org/10.3389/fnagi.2022.954090>.
- 42 Peter Schönfeld and Georg Reiser, “Why Does Brain Metabolism Not Favor Burning of Fatty Acids to Provide Energy? Reflections on Disadvantages of the Use of Free Fatty Acids as Fuel for Brain,” *Journal of Cerebral Blood Flow and Metabolism* 33, no. 10 (2013): 1493–9, <https://doi.org/10.1038/jcbfm.2013.128>.
- 43 Amick et al., “Plasma Glycocholic Acid.”
- 44 Hau D. Le et al. “The Essentiality of Arachidonic Acid and Docosahexaenoic Acid,” *Prostaglandins, Leukotrienes, and Essential Fatty Acids* 81, no. 2–3 (2009): 165–70, <https://doi.org/10.1016/j.plefa.2009.05.020>.
- 45 Ather Muneer, “The Neurobiology of Bipolar Disorder: An Integrated Approach,” *Chonnam Medical Journal* 52, no. 1 (2016): 18–37, <https://doi.org/10.4068/cmj.2016.52.1.18>.
- 46 Thomas McGrath, Richard Baskerville, Marcelo Rogero, and Linda Castell, “Emerging Evidence for the Widespread Role of Glutamatergic Dysfunction in Neuropsychiatric Diseases,” *Nutrients* 14, no. 5 (2022): 917, <https://doi.org/10.3390/nu14050917>.
- 47 David A. Kessler, *The End of Overeating* (New York, Macmillan, 2009), 18.
- 48 Ronald J. Jandacek, “Linoleic Acid: A Nutritional Quandary.” *Healthcare* 5, no. 2 (2017): 25, <https://doi.org/10.3390/healthcare5020025>.
- 49 Stephan J. Guyenet and Susan E. Carlson, “Increase in Adipose Tissue Linoleic Acid of US Adults in the Last Half Century,” *Advances in Nutrition* 6, no. 6 (2015): 660–4, <https://doi.org/10.3945/an.115.009944>.
- 50 Rowena Field, Tara Field, Fereshteh Pourkazemi, and Kieron Rooney,

“Low-Carbohydrate and Ketogenic Diets: A Scoping Review of Neurological and Inflammatory Outcomes in Human Studies and Their Relevance to Chronic Pain,” *Nutrition Research Reviews* (2022): 1–25, <https://doi.org/10.1017/S0954422422000087>.

- 51 Krasimira Aleksandrova, Liselot Koelman, and Cae Egea Rodrigues, “Dietary Patterns and Biomarkers of Oxidative Stress and Inflammation: A Systematic Review of Observational and Intervention Studies,” *Redox Biology* 42 (2021): 101869, <https://doi.org/10.1016/j.redox.2021.101869>; Omar Ramos-Lopez, Diego Martinez-Urbistondo, Juan A. Vargas-Nuñez, and J. Alfredo Martinez, “The Role of Nutrition on Meta-Inflammation: Insights and Potential Targets in Communicable and Chronic Disease Management,” *Current Obesity Reports* 11, no. 4 (2022): 305–35, <https://doi.org/10.1007/s13679-022-00490-0>.

Chapter 7

- [1](#) A. J. Verberne, W. S. Korim, A. Sabetghadam, and I. J. Llewellyn-Smith, “Adrenaline: Insights into Its Metabolic Roles in Hypoglycaemia and Diabetes,” *British Journal of Pharmacology* 173, no. 9 (2016): 1425–37, <https://doi.org/10.1111/bph.13458>.
- [2](#) T. W. Jones et al., “Enhanced Adrenomedullary Response and Increased Susceptibility to Neuroglycopenia: Mechanisms Underlying the Adverse Effects of Sugar Ingestion in Healthy Children,” *The Journal of Pediatrics* 126, no. 2 (1995): 171–7, [https://doi.org/10.1016/s0022-3476\(95\)70541-4](https://doi.org/10.1016/s0022-3476(95)70541-4).
- [3](#) D. S. Ludwig et al., “High Glycemic Index Foods, Overeating, and Obesity,” *Pediatrics* 103, no. 3 (1999): E26, <https://doi.org/10.1542/peds.103.3.e26>.
- [4](#) David S. Ludwig, *Always Hungry?: Conquer Cravings, Retain Your Fat Cells, and Lose Weight Permanently* (New York: Grand Central Life & Style, 2016), chap. 3, EPUB.
- [5](#) Ewelina Dziurkowska and Marek Wesolowski, “Cortisol as a Biomarker of Mental Disorder Severity,” *Journal of Clinical Medicine* 10, no. 21 (2021): 5204, <https://doi.org/10.3390/jcm10215204>.
- [6](#) David J. Unwin et al., “Substantial and Sustained Improvements in Blood Pressure, Weight and Lipid Profiles from a Carbohydrate Restricted Diet: An Observational Study of Insulin Resistant Patients in Primary Care,” *International Journal of Environmental Research and Public Health* 16, no. 15 (2019): 2680, <https://doi.org/10.3390/ijerph16152680>.
- [7](#) Gary Taubes, *A Case Against Sugar* (New York: Alfred A. Knopf, 2016), 31.
- [8](#) Angela Jacques et al., “The Impact of Sugar Consumption on Stress Driven, Emotional and Addictive Behaviors,” *Neuroscience and Biobehavioral Reviews* 103 (2019): 178–99, <https://doi.org/10.1016/j.neubiorev.2019.05.021>.
- [9](#) Serge H. Ahmed, Karine Guillem, and Youna Vandaele, “Sugar Addiction: Pushing the Drug-Sugar Analogy to the Limit,” *Current Opinion in Clinical Nutrition and Metabolic Care* 16, no. 4 (2013): 434–

- 9, <https://doi.org/10.1097/MCO.0b013e328361c8b8>.
- 10 Marina Brito Campos, Ida Helena Carvalho Francescantonio Menezes, Maria do Rosário Gondim Peixoto, Raquel Machado Schincaglia, “Intuitive Eating in General Aspects of Eating Behaviors in Individuals with Obesity: Randomized Clinical Trial,” *Clinical Nutrition ESPEN* 50 (2022): 24–32, <https://doi.org/10.1016/j.clnesp.2022.06.002>.
- 11 Flint, Alan J et al. “Food-Addiction Scale Measurement in 2 Cohorts of Middle-Aged and Older Women,” *The American Journal of Clinical Nutrition* 99, no. 3 (2014): 578–86, <https://doi.org/10.3945/ajcn.113.068965>; paraphrased with permission.

Chapter 8

- [1](#) Centers of Disease Control and Prevention, “Prevalence of Prediabetes Among Adults,” CDC, updated September 30, 2022, <https://www.cdc.gov/diabetes/data/statistics-report/prevalence-of-prediabetes.html>.
- [2](#) Centers of Disease Control and Prevention, “Prevalence of Diagnosed Diabetes,” CDC, updated September 30, 2022, <https://www.cdc.gov/diabetes/data/statistics-report/diagnosed-diabetes.html>.
- [3](#) Xiling Lin et al., “Global, Regional, and National Burden and Trend of Diabetes in 195 Countries and Territories: An Analysis from 1990 to 2025,” *Scientific Reports* 10, no. 1 (2020): 14790, <https://doi.org/10.1038/s41598-020-71908-9>.
- [4](#) Australian Diabetes Educators Association, “A Position Statement on Screening and Management of Prediabetes in Adults in Primary Care in Australia,” April 30, 2020, <https://www.adea.com.au/wp-content/uploads/2020/07/A-Position-Statement-on-Screening-and-Management-of-Prediabetes-in-Adults-in-Primary-Care-in-Australia.pdf>.
- [5](#) The Canadian Diabetes Association, “Prediabetes,” Diabetes Canada, 2018, <https://www.diabetes.ca/recently-diagnosed/prediabetes-toolkit>.
- [6](#) Kirsten Coppell et al., “What Predicts Regression from Pre-Diabetes to Normal Glucose Regulation Following a Primary Care Nurse-Delivered Dietary Intervention? A Study Protocol for a Prospective Cohort Study,” *BMJ Open* 9, no. 12 (2019): e033358, <https://doi.org/10.1136/bmjopen-2019-033358>.
- [7](#) Zoe Sherwood, “Prediabetes: Definition, Diagnostic Criteria and Management,” *Journal of Diabetes Nursing* 22, no. 3 (2018): 24, <https://diabetesonthenet.com/journal-diabetes-nursing/prediabetes-definition-diagnostic-criteria-and-management>.
- [8](#) Joseph R. Kraft, “Detection of Diabetes Mellitus *In Situ* (Occult Diabetes),” *Laboratory Medicine* 6, no. 2 (1975): 10–22, <https://doi.org/10.1093/labmed/6.2.10>.
- [9](#) Hang Xu et al., “Etiology of Metabolic Syndrome and Dietary

- Intervention,” *International Journal of Molecular Sciences* 20, no. 1 (2018): 128, <https://doi.org/10.3390/ijms20010128>.
- 10 Coppell et al., “What Predicts Regression.”
- 11 Gordon I. Smith et al., “Insulin Resistance Drives Hepatic De Novo Lipogenesis in Nonalcoholic Fatty Liver Disease,” *The Journal of Clinical Investigation* 130, no. 3 (2020): 1453–60, <https://doi.org/10.1172/JCI134165>.
- 12 Mohamed H. Ahmed and Asif Ali, “Nonalcoholic Fatty Liver Disease and Cholesterol Gallstones: Which Comes First?,” *Scandinavian Journal of Gastroenterology* 49, no. 5 (2014): 521–7, <https://doi.org/10.3109/00365521.2014.894119>.
- 13 Pedro L. Mangabeira Albernaz, “Hearing Loss, Dizziness, and Carbohydrate Metabolism,” *International Archives of Otorhinolaryngology* 20, no. 3 (2016): 261–70, <https://doi.org/10.1055/s-0035-1558450>.
- 14 Valeska Ormazabal et al., “Association Between Insulin Resistance and the Development of Cardiovascular Disease,” *Cardiovascular Diabetology* 17, no. 1 (2018): 122, <https://doi.org/10.1186/s12933-018-0762-4>.
- 15 Kornelia Kotseva et al., “EUROASPIRE IV: A European Society of Cardiology Survey on the Lifestyle, Risk Factor and Therapeutic Management of Coronary Patients from 24 European Countries,” *European Journal of Preventive Cardiology* 23, no. 6 (2016): 636–48, <https://doi.org/10.1177/2047487315569401>.
- 16 Hubert Kolb et al., “Insulin Translates Unfavourable Lifestyle into Obesity,” *BMC Medicine* 16, no. 1 (2018): 232, <https://doi.org/10.1186/s12916-018-1225-1>.
- 17 Giliola Calori et al., “Prevalence, Metabolic Features, and Prognosis of Metabolically Healthy Obese Italian Individuals: The Cremona Study,” *Diabetes Care* 34, no. 1 (2011): 210–5, <https://doi.org/10.2337/dc10-0665>.
- 18 Chung-Jyi Tsai, Michael F. Leitzmann, Walter C. Willett, and Edward L. Giovannucci, “Macronutrients and Insulin Resistance in Cholesterol Gallstone Disease,” *The American Journal of Gastroenterology* 103, no. 11 (2008): 2932–9, <https://doi.org/10.1111/j.1572-0241.2008.02189.x>.
- 19 Lisa D. Yee et al., “Metabolic Health, Insulin, and Breast Cancer: Why

- Oncologists Should Care About Insulin,” *Frontiers in Endocrinology* 11 (2020): 58, <https://doi.org/10.3389/fendo.2020.00058>.
- 20 Paolo Giovanni Vigneri et al., “The Insulin/IGF System in Colorectal Cancer Development and Resistance to Therapy,” *Frontiers in Oncology* 5 (2015): 230, <https://doi.org/10.3389/fonc.2015.00230>.
- 21 John C. Marshall and Andrea Dunaif. “Should All Women with PCOS be Treated for Insulin Resistance?,” *Fertility and Sterility* 97, no. 1 (2012): 18–22, <https://doi.org/10.1016/j.fertnstert.2011.11.036>.
- 22 Wolfgang Kopp, “Diet-Induced Hyperinsulinemia as a Key Factor in the Etiology of Both Benign Prostatic Hyperplasia and Essential Hypertension?,” *Nutrition and Metabolic Insights* 11 (2018): 1178638818773072, <https://doi.org/10.1177/1178638818773072>; J. Hammarsten and B. Högstedt, “Hyperinsulinaemia as a Risk Factor for Developing Benign Prostatic Hyperplasia,” *European Urology* 39, no. 2 (2001): 151–8, <https://doi.org/10.1159/000052430>.
- 23 Rajeev Sood et al., “The Correlation Between Erectile Dysfunction and Metabolic Syndrome in an Indian Population: A Cross-Sectional Observational Study,” *Arab Journal of Urology* 17, no. 3 (2019): 221–7, <https://doi.org/10.1080/2090598X.2019.1600990>.
- 24 Peng-Fei Ding, “Insulin Resistance in Ischemic Stroke: Mechanisms and Therapeutic Approaches,” *Frontiers in Endocrinology* 13 (2022): 1092431, <https://doi.org/10.3389/fendo.2022.1092431>.
- 25 David Unwin et al., “Substantial and Sustained Improvements in Blood Pressure, Weight and Lipid Profiles from a Carbohydrate Restricted Diet: An Observational Study of Insulin Resistant Patients in Primary Care,” *International Journal of Environmental Research and Public Health* 16, no. 15 (2019): 2680, <https://doi.org/10.3390/ijerph16152680>.
- 26 Ming-Sheng Zhou, Aimei Wang, and Hong Yu, “Link Between Insulin Resistance and Hypertension: What Is the Evidence from Evolutionary Biology?,” *Diabetology & Metabolic Syndrome* 6, no. 1 (2014): 12, <https://doi.org/10.1186/1758-5996-6-12>.
- 27 Nazik H. Hasrat and Asaad Q. Al-Yassen, “The Relationship Between Acne Vulgaris and Insulin Resistance,” *Cureus* 15, no. 1 (2023): e34241, <https://doi.org/10.7759/cureus.34241>.
- 28 Hiroyuki Sagesaka et al., “Type 2 Diabetes: When Does It Start?,” *Journal of the Endocrine Society* 2, no. 5 (2018): 476–484,

- [https://doi.org/10.1210/js.2018-00071.](https://doi.org/10.1210/js.2018-00071)
- 29 Suzanne M. de la Monte, “Type 3 Diabetes Is Sporadic Alzheimer’s Disease: Mini-Review.” *European Neuropsychopharmacology* 24, no. 12 (2014): 1954–60, <https://doi.org/10.1016/j.euroneuro.2014.06.008>.
- 30 “Dementia,” World Health Organization, updated September 21, 2020, <https://www.who.int/news-room/fact-sheets/detail/dementia>.
- 31 “Dementia,” World Health Organization.
- 32 Alzheimer’s Association, “2017 Alzheimer’s Disease Facts and Figures,” *Alzheimer’s Dementia* 13 (2017): 325–73, <https://www.alz.org/media/images/2017-facts-and-figures.pdf>.
- 33 Zaven S. Khachaturian, “40 Years of Alzheimer’s Research Failure: Now What?,” MedPage Today, September 13, 2018, <https://www.medpagetoday.com/neurology/alzheimersdisease/75075>.
- 34 Valentin K. Gribkoff and Leonard K. Kaczmarek, “The Need for New Approaches in CNS Drug Discovery: Why Drugs Have Failed, and What Can Be Done to Improve Outcomes,” *Neuropharmacology* 120 (2017): 11–9, <https://doi.org/10.1016/j.neuropharm.2016.03.021>; Julie Steenhuyzen, “Roche Shuttles Most Trials of Alzheimer’s Drug After Failed Trials,” Reuters, December 1, 2022, <https://www.reuters.com/business/healthcare-pharmaceuticals/roche-shuttles-most-trials-alzheimers-drug-after-failed-trials-2022-12-01/>.
- 35 “Pfizer Ends Research for New Alzheimer’s, Parkinson’s Drugs,” Reuters, January 7, 2018, <https://www.reuters.com/article/us-pfizer-alzheimers/pfizer-ends-research-for-new-alzheimers-parkinsons-drugs-idUSKBN1EW0TN>.
- 36 Jennifer Couzin-Frankel and Charles Piller, “As Some Hail New Antibody Treatment for Alzheimer’s, Safety and Benefit Questions Persist,” *Science*, 378, no. 6624 (2022): 1030–1, <https://doi.org/10.1126/science.adg0718>; Christopher H. van Dyck et al., “Lecanemab in Early Alzheimer’s Disease,” *The New England Journal of Medicine* 388, no. 1 (2023): 9–21, <https://doi.org/10.1056/NEJMoa2212948>.
- 37 George J. Brewer, “Alzheimer’s Disease Causation by Copper Toxicity and Treatment with Zinc. *Frontiers in Aging Neuroscience* 6 (2014): 92, <https://doi.org/10.3389/fnagi.2014.00092>.
- 38 Hanns Hippius and Gabriele Neundörfer, “The Discovery of

- Alzheimer's Disease," *Dialogues in Clinical Neuroscience* 5, no. 1 (2003): 101–8, <https://doi.org/10.31887/DCNS.2003.5.1/hhippius>.
- 39 Alice Park, "80% of People Think Alzheimer's Is a Normal Part of Aging," TIME. June 19, 2014, <http://time.com/2897084/80-of-people-mistakenly-think-alzheimers-is-a-normal-part-of-aging>.
- 40 Eric Steen et al., "Impaired Insulin and Insulin-Like Growth Factor Expression and Signaling Mechanisms in Alzheimer's Disease: Is This Type 3 Diabetes?," *Journal of Alzheimer's Disease* 7 no. 1 (2005): 6380, <https://doi.org/10.3233/JAD-2005-7107>.
- 41 Juliette Janson et al., "Increased Risk of Type 2 Diabetes in Alzheimer Disease," *Diabetes* 53, no. 2 (2004): 474–81, <https://doi.org/10.2337/diabetes.53.2.474>.
- 42 Claudio Barbiellini Amidei et al., "Association Between Age at Diabetes Onset and Subsequent Risk of Dementia," *JAMA* 325, no. 16 (2021): 1640–9, <https://doi.org/10.1001/jama.2021.4001>.
- 43 Sarah M. Gray, Rick I. Meijer, and Eugene J. Barrett, "Insulin Regulates Brain Function, But How Does It Get There?," *Diabetes* 63, no. 12 (2014): 3992–7, <https://doi.org/10.2337/db14-0340>.
- 44 Auriel A. Willette et al., "Association of Insulin Resistance with Cerebral Glucose Uptake in Late Middle-Aged Adults at Risk for Alzheimer Disease," *JAMA Neurology* 72, no. 9 (2015): 1013–20, <https://doi.org/10.1001/jamaneurol.2015.0613>.
- 45 Cheng-Che Lee, Chiung-Chun Huang, and Kuei-Sen Hsu, "Insulin Promotes Dendritic Spine and Synapse Formation by the PI3K/Akt/mTOR and Rac1 Signaling Pathways," *Neuropharmacology* 61, no. 4 (2011): 867–79, <https://doi.org/10.1016/j.neuropharm.2011.06.003>.
- 46 Muhammad Syahrul, Anwar Zainuddin, and Sandrine Thuret, "Nutrition, Adult Hippocampal Neurogenesis and Mental Health," *British Medical Bulletin* 103, no. 1 (2012): 89–114, <https://doi.org/10.1093/bmb/lds021>.
- 47 C. A. Grillo, G. G. Piroli, R. M. Hendry, and L. P. Reagan, "Insulin-Stimulated Translocation of GLUT4 to the Plasma Membrane in Rat Hippocampus Is PI3-Kinase Dependent," *Brain Research* 1296 (2009): 35–45, <https://doi.org/10.1016/j.brainres.2009.08.005>.
- 48 A. T. Du et al., "Magnetic Resonance Imaging of the Entorhinal Cortex

- and Hippocampus in Mild Cognitive Impairment and Alzheimer’s Disease,” *Journal of Neurology, Neurosurgery, and Psychiatry* 71, no. 4 (2001): 441–7, <https://doi.org/10.1136/jnnp.71.4.441>.
- 49 Hippius and Neundörfer, “The Discovery of Alzheimer’s Disease.”
- 50 For questioning of tau hypothesis, see: Catherine M. Cowan and Amrit Mudher, “Are Tau Aggregates Toxic or Protective in Tauopathies?” *Frontiers in Neurology* 4, (2013): 114, <https://doi.org/10.3389/fneur.2013.00114>; for questioning the amyloid hypothesis, see: Karl Herrup, “The Case for Rejecting the Amyloid Cascade Hypothesis,” *Nature Neuroscience* 18, no. 6 (2015): 794–9, <https://doi.org/10.1038/nn.4017>.
- 51 Rosebud O. Roberts et al., “Prevalence and Outcomes of Amyloid Positivity Among Persons Without Dementia in a Longitudinal, Population-Based Setting,” *JAMA Neurology* 75, no. 8 (2018): 970–9, <https://doi.org/10.1001/jamaneurol.2018.0629>.
- 52 Sy Mukherjee, “Alzheimer’s: A Trail of Disappointment for Big Pharma,” Fortune, January 18, 2019, <https://fortune.com/2019/01/18/alzheimers-a-trail-of-disappointment-for-big-pharma/>; Roberts et al., “Prevalence and Outcomes of Amyloid.”
- 53 Zenghui Wei, Jagadish Koya, and Sandra E. Reznik, “Insulin Resistance Exacerbates Alzheimer Disease via Multiple Mechanisms,” *Frontiers in Neuroscience* 15 (2021), <https://doi.org/10.3389/fnins.2021.687157>.
- 54 Ying Yang and Jian-Zhi Wang. “Nature of Tau-Associated Neurodegeneration and the Molecular Mechanisms,” *Journal of Alzheimer’s Disease* 62, no. 3 (2018): 1305–17, <https://doi.org/10.3233/JAD-170788>.
- 55 Matthew R. Brier et al., “Tau and A β Imaging, CSF Measures, and Cognition in Alzheimer’s Disease,” *Science Translational Medicine* 8, no. 338 (2016): 338ra66, <https://doi.org/10.1126/scitranslmed.aaf2362>.
- 56 Miranda E. Orr, A. Campbell Sullivan, and Bes Frost, “A Brief Overview of Tauopathy: Causes, Consequences, and Therapeutic Strategies,” *Trends in Pharmacological Sciences* 38, no. 7 (2017): 637–48, <https://doi.org/10.1016/j.tips.2017.03.011>.
- 57 Gina M. Broom, Ian C. Shaw, and Julia J. Ruckridge, “The Ketogenic Diet as a Potential Treatment and Prevention Strategy for Alzheimer’s Disease,” *Nutrition* 60 (2019): 118–121,

- [https://doi.org/10.1016/j.nut.2018.10.003.](https://doi.org/10.1016/j.nut.2018.10.003)
- 58 Harald Hampel et al., “The Cholinergic System in the Pathophysiology and Treatment of Alzheimer’s Disease,” *Brain: A Journal of Neurology* 141, no. 7 (2018): 1917–33, <https://doi.org/10.1093/brain/awy132>.
- 59 Kedar Batkulwar et al., “Advanced Glycation End Products Modulate Amyloidogenic APP Processing and Tau Phosphorylation: A Mechanistic Link between Glycation and the Development of Alzheimer’s Disease,” *ACS Chemical Neuroscience* 9, no. 5 (2018): 988–1000, <https://doi.org/10.1021/acscchemneuro.7b00410>.
- 60 Peter J. Fried, Alvaro Pascual-Leone, and Nicolas R. Bolo, “Diabetes and the Link Between Neuroplasticity and Glutamate in the Aging Human Motor Cortex,” *Clinical Neurophysiology* 130, no. 9 (2019): 1502–1510, <https://doi.org/10.1016/j.clinph.2019.04.721>.
- 61 Rui Wang and P. Hemachandra Reddy, “Role of Glutamate and NMDA Receptors in Alzheimer’s Disease,” *Journal of Alzheimer’s Disease* 57, no. 4 (2017): 1041–8, <https://doi.org/10.3233/JAD-160763>.
- 62 Leif Hertz, Ye Chen, and Helle S. Waagepetersen, “Effects of Ketone Bodies in Alzheimer’s Disease in Relation to Neural Hypometabolism, B-Amyloid Toxicity, and Astrocyte Function,” *Journal of Neurochemistry* 134, no. 1 (2015): 7–20, <https://doi.org/10.1111/jnc.13107>.
- 63 Willette et al., “Association of Insulin Resistance.”
- 64 Stephan C. Cunnane et al., “Can Ketones Compensate for Deteriorating Brain Glucose Uptake During Aging? Implications for the Risk and Treatment of Alzheimer’s Disease,” *Annals of the New York Academy of Sciences* 1367, no. 1 (2016): 12–20, <https://doi.org/10.1111/nyas.12999>.
- 65 Cunnane et al., “Can Ketones Compensate.”
- 66 Reisa A. Sperling et al., “Toward Defining the Preclinical Stages of Alzheimer’s Disease: Recommendations from the National Institute on Aging-Alzheimer’s Association Workgroups on Diagnostic Guidelines for Alzheimer’s Disease,” *Alzheimer’s & Dementia* 7, no. 3 (2011): 280–92, <https://doi.org/10.1016/j.jalz.2011.03.003>.
- 67 Tyler C. Hammond and Ai-Ling Lin, “Glucose Metabolism Is a Better Marker for Predicting Clinical Alzheimer’s Disease than Amyloid or Tau,” *Journal of Cellular Immunology* 4, no. 1 (2022): 15–18, <https://doi.org/10.33696/immunology.4.128>.

- 68 Kathleen T. Watson et al., “Incident Major Depressive Disorder Predicted by Three Measures of Insulin Resistance: A Dutch Cohort Study,” *The American Journal of Psychiatry* 178, no. 10 (2021): 914–920, <https://doi.org/10.1176/appi.ajp.2021.20101479>.
- 69 Klara Coello et al., “Metabolic Profile in Patients with Newly Diagnosed Bipolar Disorder and Their Unaffected First-Degree Relatives,” *International Journal of Bipolar Disorders* 7, no. 1 (2019): 8, <https://doi.org/10.1186/s40345-019-0142-3>.
- 70 Jakub Tomasik et al., “Association of Insulin Resistance with Schizophrenia Polygenic Risk Score and Response to Antipsychotic Treatment,” *JAMA Psychiatry* 76, no. 8 (2019): 864–867, <https://doi.org/10.1001/jamapsychiatry.2019.0304>.
- 71 Cheng-Ta Li et al., “Prefrontal Glucose Metabolism in Medication-Resistant Major Depression,” *The British Journal of Psychiatry* 206, no. 4 (2015): 316–23, <https://doi.org/10.1192/bjp.bp.113.140434>.
- 72 L. Schmaal et al., “Subcortical Brain Alterations in Major Depressive Disorder: Findings from the ENIGMA Major Depressive Disorder Working Group,” *Molecular Psychiatry* 21, no. 6 (2016): 806–12, <https://doi.org/10.1038/mp.2015.69>.
- 73 Holly Elser et al., “Association of Early-, Middle-, and Late-Life Depression with Incident Dementia in a Danish Cohort,” *JAMA Neurology* (2023): e232309, <https://doi.org/10.1001/jamaneurol.2023.2309>.
- 74 Natalie L. Rasgon and Heather A. Kenna, “Insulin Resistance in Depressive Disorders and Alzheimer’s Disease: Revisiting the Missing Link Hypothesis,” *Neurobiology of Aging* 26, Suppl 1 (2005): 103–7, <https://doi.org/10.1016/j.neurobiolaging.2005.09.004>.
- 75 Chujun Wu et al., “Cerebral Glucose Metabolism in Bipolar Disorder: A Voxel-Based Meta-Analysis of Positron Emission Tomography Studies,” *Brain and Behavior* 11, no. 5 (2021): e02117, <https://doi.org/10.1002/brb3.2117>.
- 76 Unn K. Haukvik et al., “In Vivo Hippocampal Subfield Volumes in Bipolar Disorder: A Mega-Analysis from The Enhancing Neuro Imaging Genetics through Meta-Analysis Bipolar Disorder Working Group,” *Human Brain Mapping* 43, no. 1 (2022): 385–398, <https://doi.org/10.1002/hbm.25249>.

- 77 Breno S. Diniz et al., “History of Bipolar Disorder and the Risk of Dementia: A Systematic Review and Meta-Analysis,” *The American Journal of Geriatric Psychiatry* 25, no. 4 (2017): 357–362, <https://doi.org/10.1016/j.jagp.2016.11.014>.
- 78 S. Andrea Wijtenburg et al., “Brain Insulin Resistance and Altered Brain Glucose are Related to Memory Impairments in Schizophrenia,” *Schizophrenia Research* 208 (2019): 324–330, <https://doi.org/10.1016/j.schres.2019.01.031>.
- 79 Unn K. Haukvik, Christian K. Tamnes, Erik Söderman, and Ingrid Agartz, “Neuroimaging Hippocampal Subfields in Schizophrenia and Bipolar Disorder: A Systematic Review and Meta-Analysis,” *Journal of Psychiatric Research* 104 (2018): 217–226, <https://doi.org/10.1016/j.jpsychires.2018.08.012>.
- 80 Sara El Miniawi, Vasiliki Orgeta, and Jean Stafford, “Non-Affective Psychotic Disorders and Risk of Dementia: A Systematic Review and Meta-Analysis,” *Psychological Medicine*, 52, no. 15 (2022): 1–13, <https://doi.org/10.1017/S0033291722002781>.
- 81 A. J. Zametkin et al., “Cerebral Glucose Metabolism in Adults with Hyperactivity of Childhood Onset,” *The New England Journal of Medicine* 323, no. 20 (1990): 1361–6, <https://doi.org/10.1056/NEJM199011153232001>.
- 82 J. M. De La Fuente et al., “Brain Glucose Metabolism in Borderline Personality Disorder,” *Journal of Psychiatric Research* 31, no. 5 (1997): 531–41, [https://doi.org/10.1016/s0022-3956\(97\)00001-0](https://doi.org/10.1016/s0022-3956(97)00001-0).
- 83 Sanjaya Saxena et al., “Cerebral Glucose Metabolism in Obsessive-Compulsive Hoarding,” *The American Journal of Psychiatry* 161, no. 6 (2004): 1038–48, <https://doi.org/10.1176/appi.ajp.161.6.1038>.
- 84 Cynthia V. Calkin and Martin Alda, “Insulin Resistance in Bipolar Disorder: Relevance to Routine Clinical Care,” *Bipolar Disorders* 17, no. 6 (2015): 683–8, <https://doi.org/10.1111/bdi.12330>.
- 85 Cynthia V. Calkin et al., “Treating Insulin Resistance with Metformin as a Strategy to Improve Clinical Outcomes in Treatment-Resistant Bipolar Depression (the TRIO-BD Study): A Randomized, Quadruple-Masked, Placebo-Controlled Clinical Trial,” *The Journal of Clinical Psychiatry* 83, no. 2 (2022): 21m14022, <https://doi.org/10.4088/JCP.21m14022>.
- 86 Cynthia Calkin, email message to author, February 25, 2023.

- 87 Jon Tattrie, “50-Year-Old Diabetes Drug Helps Patients with Bipolar Disorder, Study Finds,” CBC News, July 18, 2022, <https://www.cbc.ca/news/canada/nova-scotia/50-year-old-diabetes-drug-helps-patients-with-bipolar-disorder-study-finds-1.6524070>.
- 88 Auriel A. Willette et al., “Insulin Resistance Predicts Brain Amyloid Deposition in Late Middle-Aged Adults,” *Alzheimer’s & Dementia*. 11, no. 5 (2015): 504–10.e1, <https://doi.org/10.1016/j.jalz.2014.03.011>.

Chapter 9

- [1](#) Albert Danan, Eric C. Westman, Laura R. Saslow, and Georgia Ede, “The Ketogenic Diet for Refractory Mental Illness: A Retrospective Analysis of 31 Inpatients,” *Frontiers in Psychiatry* 13, 951376 (2022), <https://doi.org/10.3389/fpsyg.2022.951376>.
- [2](#) Sookyong Koh, Nina Dupuis, and Stéphane Auvin, “Ketogenic Diet and Neuroinflammation,” *Epilepsy Research* 167 (2020): 106454, <https://doi.org/10.1016/j.eplepsyres.2020.106454>.
- [3](#) Antonio Napolitano et al., “The Ketogenic Diet Increases In Vivo Glutathione Levels in Patients with Epilepsy,” *Metabolites* 10, no. 12 (2020): 504, <https://doi.org/10.3390/metabo10120504>.
- [4](#) Richard E. Frye, “Mitochondrial Dysfunction in Autism Spectrum Disorder: Unique Abnormalities and Targeted Treatments,” *Seminars in Pediatric Neurology* 35, (October 2020): 100829, <https://doi.org/10.1016/j.spen.2020.100829>.
- [5](#) Frye, “Mitochondrial Dysfunction.”
- [6](#) Krisztina Marosi et al., “3-Hydroxybutyrate Regulates Energy Metabolism and Induces BDNF Expression in Cerebral Cortical Neurons,” *Journal of Neurochemistry* 139, no. 5 (2016): 769–81, <https://doi.org/10.1111/jnc.13868>.
- [7](#) Zoltána Sarnyai, Ann-Katrina Kraeuter, and Christopher M. Palmer, “Ketogenic Diet for Schizophrenia: Clinical Implication,” *Current Opinion in Psychiatry* 32, no. 5 (2019): 394–401, <https://doi.org/10.1097/YCO.0000000000000535>.
- [8](#) Alessandro Ricci, Maia A. Idzikowski, Claudio N. Soares, and Elisa Brietzke, “Exploring the Mechanisms of Action of the Antidepressant Effect of the Ketogenic Diet,” *Reviews in the Neurosciences* 31, no. 6 (2020): 637–648, <https://doi.org/10.1515/revneuro-2019-0073>.
- [9](#) Lilianne R. Mujica-Parodi et al., “Diet Modulates Brain Network Stability, a Biomarker for Brain Aging, in Young Adults,” *Proceedings of the National Academy of Sciences of the United States of America* 117, no. 11 (2020): 6170–7, <https://doi.org/10.1073/pnas.1913042117>.
- [10](#) Jeffrey L. B. Bohnen, et al., “Ketogenic-Mimicking Diet as a Therapeutic Modality for Bipolar Disorder: Biomechanistic Rationale

- and Protocol for a Pilot Clinical Trial,” *Nutrients* 15, no. 13 (2023): 3068, <https://doi.org/10.3390/nu15133068>.
- 11 Alexandre Courchesne-Loyer et al., “Inverse Relationship between Brain Glucose and Ketone Metabolism in Adults during Short-Term Moderate Dietary Ketosis: A Dual Tracer Quantitative Positron Emission Tomography Study,” *Journal of Cerebral Blood Flow & Metabolism* 37, no. 7 (2017): 2485–2493, <https://doi.org/10.1177/0271678X16669366>.
- 12 Zoe Harcombe, “The Science of Reversing Type 2 Diabetes with Low Carbohydrates,” presentation to the All-Party Parliamentary Group for Diabetes, UK Parliament, February 6, 2019, YouTube, 12:12, <https://www.youtube.com/watch?v=lQVsHtPUUQI>.
- 13 Iain Campbell and Harry Campbell, “Mechanisms of Insulin Resistance, Mitochondrial Dysfunction and the Action of the Ketogenic Diet in Bipolar Disorder: Focus on the PI3K/AKT/HIF1-a Pathway,” *Medical Hypotheses* 145 (2020): 110299, <https://doi.org/10.1016/j.mehy.2020.110299>.
- 14 James R. Phelps, Susan V. Siemers, and Rif S. El-Mallakh, “The Ketogenic Diet for Type II Bipolar Disorder,” *Neurocase* 19, no. 5 (2013): 423–6, <https://doi.org/10.1080/13554794.2012.690421>; Michael Saraga, Nicole Misson, and Elaine Cattani, “Ketogenic Diet in Bipolar Disorder,” *Bipolar Disorder* 22, no. 7 (2020): 765, <https://doi.org/10.1111/bdi.13013>.
- 15 Rebecca N. Adams et al., “Depressive Symptoms Improve over 2 Years of Type 2 Diabetes Treatment Via a Digital Continuous Remote Care Intervention Focused on Carbohydrate Restriction,” *Journal of Behavioral Medicine* 45, no. 3 (2022): 416–27, <https://doi.org/10.1007/s10865-021-00272-4>.
- 16 Gerwyn Morris et al., “The Role of Microglia in Neuroprogressive Disorders: Mechanisms and Possible Neurotherapeutic Effects of Induced Ketosis.” *Progress in Neuro-psychopharmacology & Biological Psychiatry* 99 (2020): 109858, <https://doi.org/10.1016/j.pnpbp.2020.109858>.
- 17 Abel Pacheco, W. S. Easterling, and Margaret W. Pryer, “A Pilot Study of the Ketogenic Diet in Schizophrenia,” *American Journal of Psychiatry* 121 (1965): 1110–1, <https://doi.org/10.1176/ajp.121.11.1110>.
- 18 Bryan D. Kraft and Eric C. Westman, “Schizophrenia, Gluten, and Low-

- Carbohydrate, Ketogenic Diets: A Case Report and Review of the Literature,” *Nutrition & Metabolism* 6 (2009): 10, <https://doi.org/10.1186/1743-7075-6-10>.
- 19 Christopher M. Palmer, “Ketogenic Diet in the Treatment of Schizoaffective Disorder: Two Case Studies,” *Schizophrenia Research* 189 (2017): 208–9, <https://doi.org/10.1016/j.schres.2017.01.053>; Javier Gilbert-Jaramillo et al., “The Effects of the Ketogenic Diet on Psychiatric Symptomatology, Weight and Metabolic Dysfunction in Schizophrenia Patients,” *Clinical Nutrition and Metabolism* 1, no. 1 (2018): 1–5, <https://doi.org/10.15761/CNM.1000105>; Christopher M. Palmer, Javier Gilbert-Jaramillo, and Eric C. Westman, “The Ketogenic Diet and Remission of Psychotic Symptoms in Schizophrenia: Two Case Studies,” *Schizophrenia Research* 208 (2019): 439–40, <https://doi.org/10.1016/j.schres.2019.03.019>.
- 20 Zoltán Sarnyai, Ann-Katrin Kraeuter, and Christopher M. Palmer, “Ketogenic Diet for Schizophrenia: Clinical Implication,” *Current Opinion in Psychiatry* 32, no. 5 (2019): 394–401, <https://doi.org/10.1097/YCO.0000000000000535>.
- 21 Jinan Zeidan et al., “Global Prevalence of Autism: A Systematic Review Update,” *Autism Research* 15, no. 5 (2022): 778–90, <https://doi.org/10.1002/aur.2696>.
- 22 Richard E. Frye, “Mitochondrial Dysfunction in Autism Spectrum Disorder: Unique Abnormalities and Targeted Treatments,” *Seminars in Pediatric Neurology* 35 (2020): 100829, <https://doi.org/10.1016/j.spen.2020.100829>.
- 23 Matthew Carmen et al., “Treating Binge Eating and Food Addiction Symptoms with Low-Carbohydrate Ketogenic Diets: A Case Series,” *Journal of Eating Disorders* 8 (2020): 2, <https://doi.org/10.1186/s40337-020-0278-7>.
- 24 Shebani Sethi Dalai, Anika Sinha, and Ashley N. Gearhardt, “Low Carbohydrate Ketogenic Therapy as a Metabolic Treatment for Binge Eating and Ultraprocessed Food Addiction,” *Current Opinion in Endocrinology, Diabetes, and Obesity* 27, no. 5 (2020): 275–82, <https://doi.org/10.1097/MED.0000000000000571>.
- 25 Corinde E. Wiers et al., “Ketogenic Diet Reduces Alcohol Withdrawal Symptoms in Humans and Alcohol Intake in Rodents,” *Science*

Advances 7, no. 15 (2021): eabf6780,
<https://doi.org/10.1126/sciadv.abf6780>.

- 26 Robert Krikorian et al., “Dietary Ketosis Enhances Memory in Mild Cognitive Impairment,” *Neurobiology of Aging* 33, no. 2 (2012): 425.e19-27, <https://doi.org/10.1016/j.neurobiolaging.2010.10.006>.
- 27 Matthew K. Taylor et al., “Feasibility and Efficacy Data from a Ketogenic Diet Intervention in Alzheimer’s Disease,” *Alzheimer’s & Dementia* 4 (2017): 28–36, <https://doi.org/10.1016/j.jalz.2017.11.002>.
- 28 Matthew C. L. Phillips et al., “Randomized Crossover Trial of a Modified Ketogenic Diet in Alzheimer’s Disease,” *Alzheimer’s Research & Therapy* 13, no. 1 (2021): 51, <https://doi.org/10.1186/s13195-021-00783-x>.

Chapter 10

- [1](#) G. Lombardi-Boccia, B. Martinez-Dominguez, and A. Aguzzi, “Total Heme and Non-heme Iron in Raw and Cooked Meats,” *Journal of Food Science*, 67, (2002): 1738–41, <https://doi.org/10.1111/j.1365-2621.2002.tb08715.x>.
- [2](#) Briana Pobiner, “Meat-Eating Among Earliest Humans,” *American Scientist* 104, no. 2 (March-April 2016): 110–7, <https://doi.org/10.1511/2016.119.110>.
- [3](#) Sergio Almécija et al., “Fossil Apes and Human Evolution,” *Science (New York, N.Y.)* 372, no. 6542 (2021): eabb4363, <https://doi.org/10.1126/science.abb4363>.
- [4](#) Joseph V. Ferraro et al., “Earliest Archaeological Evidence of Persistent Hominin Carnivory,” *PLoS ONE* 8, no. 4 (2013): e62174, <https://doi.org/10.1371/journal.pone.0062174>.
- [5](#) Pobiner, “Meat-Eating.”
- [6](#) Katherine Milton, “Nutritional Characteristics of Wild Primate Foods: Do the Diets of Our Closest Living Relatives Have Lessons for Us?” *Nutrition* 15, no. 6 (1999): 488–98, [https://doi.org/10.1016/s0899-9007\(99\)00078-7](https://doi.org/10.1016/s0899-9007(99)00078-7).
- [7](#) Felipe Mora-Bermúdez et al., “Differences and Similarities Between Human and Chimpanzee Neural Progenitors During Cerebral Cortex Development,” *eLife* 5 (2016): e18683, <https://doi.org/10.7554/eLife.18683>.
- [8](#) Leslie C. Aiello and Peter Wheeler, “The Expensive-Tissue Hypothesis: The Brain and the Digestive System in Human and Primate Evolution,” *Current Anthropology* 36, no. 2 (1995): 199–221, <http://www.jstor.org/stable/2744104>.
- [9](#) Katharine Milton, “The Critical Role Played by Animal Source Foods in Human (*Homo*) Evolution,” *The Journal of Nutrition* 133, no. 11 Suppl 2 (2003): 3886S–3892S, <https://doi.org/10.1093/jn/133.11.3886S>.
- [10](#) Alida Melse-Boonstra, “Bioavailability of Micronutrients from Nutrient-Dense Whole Foods: Zooming in on Dairy, Vegetables, and Fruits,” *Frontiers in Nutrition* 7, no. 101 (2020): 101, <https://doi.org/10.3389/fnut.2020.00101>.

- 11 Victor E. Levine, “The Value of Meat as an Antiscorbutic,” *The American Journal of Digestive Diseases* 8 (1941): 454–63, <https://doi.org/10.1007/BF03014680>.
- 12 Amber O’Hearn, “Can a Carnivore Diet Provide All Essential Nutrients?” *Current Opinion in Endocrinology, Diabetes, and Obesity* 27, no. 5 (2020): 312–6, <https://doi.org/10.1097/MED.0000000000000576>.
- 13 Institute of Medicine. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. (Washington, DC: The National Academies Press, 2005), <https://doi.org/10.17226/10490>.
- 14 Ludovico Alisi et al., “The Relationships Between Vitamin K and Cognition: A Review of Current Evidence.” *Frontiers in Neurology* 10 (2019): 239, <https://doi.org/10.3389/fneur.2019.00239>.
- 15 Institute of Medicine. *Dietary Reference Intakes*. As retinol activity equivalents (RAEs) 1 RAE = 1 µg retinol, 12 µg β-carotene, 24 µg α-carotene, or 24 µg β-cryptoxanthin. The RAE for dietary provitamin A carotenoids is twofold greater than retinol equivalents (RE), whereas the RAE for preformed vitamin A is the same as RE.
- 16 K. L. Beck, “Anemia: Prevention and Dietary Strategies,” in *Encyclopedia of Food and Health*, ed. Benjamin Caballero, Paul M. Finglas, and Fidel Toldrá (Academic Press, 2016), 164–8, <https://doi.org/10.1016/b978-0-12-384947-2.00030-1>.
- 17 Graham. C. Burdge and Stephen. A. Wootton, “Conversion of Alpha-Linolenic Acid to Eicosapentaenoic, Docosapentaenoic and Docosahexaenoic Acids in Young Women,” *The British Journal of Nutrition* 88, no. 4 (2002): 411–20, <https://doi.org/10.1079/BJN2002689>.
- 18 Burdge and Wootton, “Conversion of Alpha-Linolenic Acid.”
- 19 Lierre Keith, *The Vegetarian Myth*, (Crescent City, CA: Flashpoint Press, 2009), 82, 92–3.
- 20 Shila Minari Hargreaves, António Raposo, Ariana Saraiva, and Renata Puppin Zandonadi, “Vegetarian Diet: An Overview through the Perspective of Quality of Life Domains,” *International Journal of Environmental Research and Public Health* 18, no. 8 (2021): 4067, <https://doi.org/10.3390/ijerph18084067>.

- 21 Belinda Fettke, email to author, July 2, 2022.
- 22 Margaret Puskar-Pasewicz, *Cultural Encyclopedia of Vegetarianism*. (Santa Barbara, CA: Greenwood, 2012), <http://www.credoreference.com/book/abcvegetarian>.
- 23 Jim E. Banta et al., “The Global Influence of the Seventh-Day Adventist Church on Diet,” *Religions* 9, no. 9 (2018): 251, <https://doi.org/10.3390/rel9090251>.
- 24 Victor W. Zhong et al., “Associations of Processed Meat, Unprocessed Red Meat, Poultry, or Fish Intake with Incident Cardiovascular Disease and All-Cause Mortality,” *JAMA Internal Medicine* 180, no. 4 (2020 Apr 1): 503–12, <https://doi.org/10.1001/jamainternmed.2019.6969>.
- 25 D. A. Hobbs-Grimmer, D. I. Givens, and J. A. Lovegrove, “Associations Between Red Meat, Processed Red Meat and Total Red and Processed Red Meat Consumption, Nutritional Adequacy and Markers of Health and Cardio-Metabolic Diseases in British Adults: A Cross-Sectional Analysis Using Data from UK National Diet and Nutrition Survey,” *European Journal of Nutrition*, 60, no. 6 (2021): 2979–97, <https://doi.org/10.1007/s00394-021-02486-3>.
- 26 Arne Astrup et al., “Saturated Fats and Health: A Reassessment and Proposal for Food-Based Recommendations: JACC State-of-the-Art Review,” *Journal of the American College of Cardiology* 76, no. 7 (2020): 844–57, <https://doi.org/10.1016/j.jacc.2020.05.077>.
- 27 Arne Astrup et al., “Dietary Saturated Fats and Health: Are the US Guidelines Evidence-Based?” *Nutrients* 13, no. 10 (2021): 3305, <https://doi.org/10.3390/nu13103305>.
- 28 Robert A. Koeth et al., “Intestinal Microbiota Metabolism of L-Carnitine, a Nutrient in Red Meat, Promotes Atherosclerosis,” *Nature Medicine* 19, no. 5 (2013): 576–85, <https://doi.org/10.1038/nm.3145>.
- 29 Véronique Bouvard et al., “Carcinogenicity of Consumption of Red and Processed Meat,” *The Lancet. Oncology* 16, no. 16 (2015): 1599–600, [https://doi.org/10.1016/S1470-2045\(15\)00444-1](https://doi.org/10.1016/S1470-2045(15)00444-1).
- 30 Fabrice Pierre et al., “Beef Meat and Blood Sausage Promote the Formation of Azoxymethane-Induced Mucin-Depleted Foci and Aberrant Crypt Foci in Rat Colons,” *The Journal of Nutrition* 134, no. 10 (October 2004): 2711–6, <https://doi.org/10.1093/jn/134.10.2711>.
- 31 Fabrice H. F. Pierre et al., “Calcium and α-Tocopherol Suppress Cured-

Meat Promotion of Chemically Induced Colon Carcinogenesis in Rats and Reduce Associated Biomarkers in Human Volunteers,” *The American Journal of Clinical Nutrition* 98, no. 5, (2013): 1255–62, <https://doi.org/10.3945/ajcn.113.061069>.

- 32 Nadia M. Bastide, Fabrice H. F. Pierre, and Denis E. Corpet, “Heme Iron from Meat and Risk of Colorectal Cancer: A Meta-Analysis and a Review of the Mechanisms Involved,” *Cancer Prevention Research* 4, no. 2 (2011): 177–84, <https://doi.org/10.1158/1940-6207.CAPR-10-0113>; Denise Grotto et al., “Importance of the Lipid Peroxidation Biomarkers and Methodological Aspects FOR Malondialdehyde Quantification,” *Química Nova* 32, no. 1 (2009): 169–74, <https://doi.org/10.1590/S0100-40422009000100032>.
- 33 Richard K. Le Leu et al., “Butyrylated Starch Intake Can Prevent Red Meat-Induced O6-Methyl-2-Deoxyguanosine Adducts in Human Rectal Tissue: A Randomised Clinical Trial,” *The British Journal of Nutrition* 114, no. 2 (2015): 220–30, <https://doi.org/10.1017/S0007114515001750>.
- 34 Markus Christmann, Barbara Verbeek, Wynand P. Roos, and Bernd Kaina, “O⁶-Methylguanine-DNA Methyltransferase (MGMT) in Normal Tissues and Tumors: Enzyme Activity, Promoter Methylation and Immunohistochemistry,” *Biochimica et Biophysica Acta* 1816, no. 2 (2011): 179–90, <https://doi.org/10.1016/j.bbcan.2011.06.002>.
- 35 Michelle H. Lewin et al., “Red Meat Enhances the Colonic Formation of the DNA Adduct O6-Carboxymethyl Guanine: Implications for Colorectal Cancer Risk,” *Cancer Research* 66, no. 3 (2006): 1859–65, <https://doi.org/10.1158/0008-5472.CAN-05-2237>.
- 36 Pattama Senthong et al., “The Nitrosated Bile Acid DNA Lesion O6-Carboxymethylguanine Is a Substrate for the Human DNA Repair Protein O6-Methylguanine-DNA Methyltransferase,” *Nucleic Acids Research* 41, no. 5 (2013): 3047–55, <https://doi.org/10.1093/nar/gks1476>.
- 37 Marije Oostindjer et al., “The Role of Red and Processed Meat in Colorectal Cancer Development: A Perspective,” *Meat Science* 97, no. 4 (2014): 583–96, <https://doi.org/10.1016/j.meatsci.2014.02.011>.
- 38 Haley Lescinsky et al., “Health Effects Associated with Consumption of Unprocessed Red Meat: A Burden of Proof Study.” *Nature Medicine* 28, no. 10 (2022): 2075–82, <https://doi.org/10.1038/s41591-022-01968-z>;

- Vanessa L. Z. Gordon-Dseagu et al., “Troubling Assumptions Behind GBD 2019 on the Health Risks of Red Meat.” *Lancet* 400, no. 10350 (2022): 427–8, [https://doi.org/10.1016/S0140-6736\(22\)01283-1](https://doi.org/10.1016/S0140-6736(22)01283-1).
- [39](#) Jael Goldfine, “A Brief History of Impossible Foods: How ‘Bleeding’ Plant-Based Burgers Started a Food Industry Trend,” *B2: The Business of Business*, November 5, 2020, <https://www.businessofbusiness.com/articles/impossible-foods-plant-based-burgers-milk-food-industry-trend-data/>.
- [40](#) David M. Klurfeld, “Research Gaps in Evaluating the Relationship of Meat and Health,” *Meat Science* 109 (2015): 86–95, <https://doi.org/10.1016/j.meatsci.2015.05.022>.
- [41](#) Raphaëlle L. Santarelli et al., “Meat Processing and Colon Carcinogenesis: Cooked, Nitrite-Treated, and Oxidized High-Heme Cured Meat Promotes Mucin-Depleted Foci in Rats,” *Cancer Prevention Research (Philadelphia, PA)* 3, no. 7 (July 2010): 852–64, <https://doi.org/10.1158/1940-6207.CAPR-09-0160>.
- [42](#) Raphaëlle L. Santarelli et al., “Meat Processing and Colon Carcinogenesis: Cooked, Nitrite-Treated, and Oxidized High-Heme Cured Meat Promotes Mucin-Depleted Foci in Rats,” *Cancer Prevention Research (Philadelphia, PA)* 3, no. 7 (July 2010): 852–64, <https://doi.org/10.1158/1940-6207.CAPR-09-0160>.
- [43](#) Geni Rodrigues Sampaio et al., “Polycyclic Aromatic Hydrocarbons in Foods: Biological Effects, Legislation, Occurrence, Analytical Methods, and Strategies to Reduce Their Formation,” *International Journal of Molecular Sciences* 22, no. 11 (June 2021): 6010, <https://doi.org/10.3390/ijms22116010>.
- [44](#) Norman G. Hord, Yaoping Tang, and Nathan S. Bryan, “Food Sources of Nitrates and Nitrites: The Physiologic Context for Potential Health Benefits,” *The American Journal of Clinical Nutrition* 90, no. 1 (July 2009): 1–10, <https://doi.org/10.3945/ajcn.2008.2711>.
- [45](#) Małgorzata Karwowska and Anna Kononiuk, “Nitrates/Nitrites in Food—Risk for Nitrosative Stress and Benefits,” *Antioxidants* 9, no. 3 (2020): 241, <https://doi.org/10.3390/antiox9030241>.
- [46](#) A.V. Kurpad, “Protein: Quality and Sources,” in *Encyclopedia of Human Nutrition (Third Edition)*, ed. Benjamin Caballero (Academic Press, 2013): 123–30, <https://doi.org/10.1016/B978-0-12-375083-9.00241-5>.

- 47 S. H. Holt, J. C. Miller, and P. Petocz, “An Insulin Index of Foods: The Insulin Demand Generated by 1000-kJ Portions of Common Foods,” *The American Journal of Clinical Nutrition* 66, no. 5 (1997): 1264–76, <https://doi.org/10.1093/ajcn/66.5.1264>
- 48 Marta Guasch-Ferré et al., “Meta-Analysis of Randomized Controlled Trials of Red Meat Consumption in Comparison with Various Comparison Diets on Cardiovascular Risk Factors,” *Circulation* 139, no. 15 (2019): 1828–45, <https://doi.org/10.1161/CIRCULATIONAHA.118.035225>.
- 49 David J. Unwin et al., “Substantial and Sustained Improvements in Blood Pressure, Weight and Lipid Profiles from a Carbohydrate Restricted Diet: An Observational Study of Insulin Resistant Patients in Primary Care,” *International Journal of Environmental Research and Public Health* 16, no. 15 (2019): 2680, <https://doi.org/10.3390/ijerph16152680>.
- 50 Radica Z. Alicic, Michele T. Rooney, and Katherine R. Tuttle, “Diabetic Kidney Disease: Challenges, Progress, and Possibilities,” *Clinical Journal of the American Society of Nephrology* 12, no. 12 (2017): 2032–45, <https://doi.org/10.2215/CJN.11491116>.
- 51 Walter S. McClellan and Eugene F. Du Bois, “Clinical Calorimetry XLV: Prolonged Meat Diets with a Study of Kidney Function and Ketosis,” *Journal of Biological Chemistry* 87, no. 3 (July 1930): 651–68, [https://doi.org/10.1016/s0021-9258\(18\)76842-7](https://doi.org/10.1016/s0021-9258(18)76842-7).
- 52 Jose Antonio and Anya Ellerbroek, “Case Reports on Well-Trained Bodybuilders: Two Years on a High Protein Diet,” *Journal of Exercise Physiology* 21, no. 1 (February 2018): 14–24; Jose Antonio et al., “A High Protein Diet Has No Harmful Effects: A One-Year Crossover Study in Resistance-Trained Males,” *Journal of Nutrition and Metabolism* 2016 (2016): 9104792, <https://doi.org/10.1155/2016/9104792>.
- 53 Saeid Safiri et al., “Prevalence, Incidence, and Years Lived with Disability Due to Gout and Its Attributable Risk Factors for 195 Countries and Territories 1990–2017: A Systematic Analysis of the Global Burden of Disease Study 2017,” *Arthritis & Rheumatology (Hoboken, NJ)* 72, no. 11 (2020): 1916–27, <https://doi.org/10.1002/art.41404>.

- 54 Chio Yokose, Natalie McCormick, and Hyon K. Choi, “The Role of Diet in Hyperuricemia and Gout,” *Current Opinion in Rheumatology* 33, no. 2 (2021): 135–44, <https://doi.org/10.1097/BOR.0000000000000779>.
- 55 Lauren E. O’Connor et al., “Effects of Total Red Meat Intake on Glycemic Control and Inflammatory Biomarkers: A Meta-Analysis of Randomized Controlled Trials,” *Advances in Nutrition (Bethesda, MD)* 12, no. 1 (2021): 115–27, <https://doi.org/10.1093/advances/nmaa096>.
- 56 Ferdinando Giacco and Michael Brownlee, “Oxidative Stress and Diabetic Complications,” *Circulation Research* 107 no. 9 (2010): 1058–70, <https://doi.org/10.1161/CIRCRESAHA.110.223545>.
- 57 U.S. Department of Agriculture, Agricultural Research Service. *FoodData Central*, 2019. <https://fdc.nal.usda.gov>.
- 58 Diana Rodgers and Robb Wolf, *Sacred Cow: The Case for (Better) Meat* (Dallas TX: BenBella Books, 2020).
- 59 Nicolette Hahn Niman, *Defending Beef: The Case for Sustainable Meat Production* (White River Junction, VT: Chelsea Green, 2015), 81.

Chapter 11

- [1](#) Selin Sergin, “Fatty Acid and Antioxidant Profile of Eggs from Pasture-Raised Hens Fed a Corn- and Soy-Free Diet and Supplemented with Grass-Fed Beef Suet and Liver,” *Foods* 11, no. 21 (2022): 3404, <https://doi.org/10.3390/foods11213404>.
- [2](#) E. Rochelle et al., “The Effects of 1 Egg Per Day on Iron and Anemia Status among Young Malawian Children: A Secondary Analysis of a Randomized Controlled Trial,” *Current Developments in Nutrition* 6, no. 6 (June 2022): nzac094, <https://doi.org/10.1093/cdn/nzac094>.
- [3](#) S.-I. Ishikawa, S. Tamaki, K. Arihara, and M. Itoh, “Egg Yolk Protein and Egg Yolk Phosvitin Inhibit Calcium, Magnesium, and Iron Absorptions in Rats,” *Journal of Food Science* 72, no. 6 (2007): S412–9, <https://doi.org/10.1111/j.1750-3841.2007.00417.x>.
- [4](#) L. Hallberg and L. Hulthén, “Prediction of Dietary Iron Absorption: An Algorithm for Calculating Absorption and Bioavailability of Dietary Iron,” *The American Journal of Clinical Nutrition* 71, no. 5 (2000): 1147–60, <https://doi.org/10.1093/ajcn/71.5.1147>.
- [5](#) Sophie Réhault-Godbert, Nicolas Guyot, and Yves Nys, “The Golden Egg: Nutritional Value, Bioactivities, and Emerging Benefits for Human Health,” *Nutrients* 11, no. 3 (March 2019):684, <http://doi.org/10.3390/nu11030684>.
- [6](#) Sasan Jalili-Firoozinezhad et al., “Chicken Egg White: Hatching of a New Old Biomaterial,” *Materials Today* 40 (2020): 193–214, <https://doi.org/10.1016/j.mattod.2020.05.022>; Nicolas Guyot et al., “Antibacterial Activity of Egg White: Influence of Physico-Chemical Conditions,” In *15. European Symposium on the Quality of Eggs and Egg Products, 21. European Symposium on the Quality of Poultry Meat, Bergamo, Italy, September 2013*, World’s Poultry Science Association: Italian Branch, *World’s Poultry Science Journal* 69, supplement (2013): 124, <https://hal.science/hal-01209474>.
- [7](#) I. Seuss-Balm, “Nutritional Evaluation of Egg Components,” In *XIth European Symposium on the Quality of Eggs and Egg Products, The Netherlands, May 23-26, 2005*, <https://www.cabi.org/uploads/animal-science/worlds-poultry-science-association/WPSA-the-netherlands->

- 2005/107.pdf.
- 8 Philip Mathew and Jennifer L. Pfleghaar, “Egg Allergy,” In StatPearls [Internet]. (Treasure Island, FL: StatPearls Publishing, 2022), Updated April 30, 2022, <https://www.statpearls.com/point-of-care/20931>.
- 9 Irvine H. Page et al., “Dietary Fat and Its Relation to Heart Attacks and Strokes,” *Circulation* 23, no. 1 (1961): 133–136.
- 10 US Senate Select Committee on Nutrition and Human Needs, *Dietary Goals*, 42.
- 11 US Senate Select Committee on Nutrition and Human Needs, *Dietary Goals for the United States* (Washington DC: US Government Printing Office, 1977): XXXVIII, <https://naldc.nal.usda.gov/catalog/1759572>.
- 12 US Department of Health and Human Services, *Nutrition and Your Health: Dietary Guidelines for Americans* (Washington, DC: US Department of Agriculture, 1980): 12, <https://www.dietaryguidelines.gov/about-dietary-guidelines/previous-editions/1980-dietary-guidelines-americans>.
- 13 Robert H. Eckel et al., “2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines,” *Journal of the American College of Cardiology* 63, no. 25 Pt B (2014): 2970, <https://doi.org/10.1016/j.jacc.2013.11.003>.
- 14 Dietary Guidelines Advisory Committee, “Part D Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends,” In *Scientific Report of the 2015 Dietary Guidelines Advisory Committee: Advisory Report to the Secretary of Health and Human Services and the Secretary of Agriculture* (Washington, DC: U.S. Department of Agriculture, Agricultural Research Service, 2015), 17, <https://health.gov/sites/default/files/2019-09/Scientific-Report-of-the-2015-Dietary-Guidelines-Advisory-Committee.pdf>.
- 15 Rylee T. Ahnen and Joanne L. Slavin, “Eggs as Part of a Healthy Eating Pattern,” in *Eggs as Functional Foods and Nutraceuticals for Human Health*, ed. Jianping Wu (Cambridge: Royal Society of Chemistry, 2019), <https://doi.org/10.1039/9781788013833-00001>.
- 16 Brett Molina, “An Egg a Day May Reduce Heart Disease Risk, Study Finds,” *USA Today*, May 22, 2018, <https://www.usatoday.com/story/news/nation-now/2018/05/22/eggs->

- heart-disease-risk/631778002/.
- 17 “Eggs Raise the Risk for Heart Disease and Death,” Physicians Committee for Responsible Medicine, March 18, 2019, <https://www.pcrm.org/news/health-nutrition/eggs-raise-risk-heart-disease-and-death>.
- 18 Amy Roeder, “An Egg a Day Is OK,” *Harvard Gazette*, March 4, 2020, <https://news.harvard.edu/gazette/story/2020/03/moderate-egg-consumption-gets-the-green-light-again/>.
- 19 Joe Pinkstone, “Step Away from the Omelette: Eating Just Half an Egg a Day Increases Your Risk of DEATH by 7%—Unless You Ditch the Yolks, Researchers Claim,” *Daily Mail*, February 9, 2021, <https://www.dailymail.co.uk/sciencetech/article-9241937/Eating-just-half-egg-day-increases-risk-DEATH-7.html>.
- 20 Jean-Philippe Drouin-Chartier et al., “Egg Consumption and Risk of Cardiovascular Disease: Three Large Prospective US Cohort Studies, Systematic Review, and Updated Meta-Analysis,” *BMJ* 368 (2020): m513, <https://doi.org/10.1136/bmj.m513>.
- 21 US Department of Agriculture and US Department of Health and Human Services. *Dietary Guidelines for Americans, 2020–2025*. 9th Edition. (December 2020): 44. Available at DietaryGuidelines.gov.
- 22 Uffe Ravnskov et al., “LDL-C Does Not Cause Cardiovascular Disease: A Comprehensive Review of the Current Literature,” *Expert Review of Clinical Pharmacology* 11, no. 10 (2018): 959–70, <https://doi.org/10.1080/17512433.2018.1519391>.
- 23 Michael A. Gimbrone, Jr. and Guillermo García-Cardeña, “Endothelial Cell Dysfunction and the Pathobiology of Atherosclerosis,” *Circulation Research* 118, no. 4 (2016): 620–36, <https://doi.org/10.1161/CIRCRESAHA.115.306301>.
- 24 Amit Sachdeva et al., “Lipid Levels in Patients Hospitalized with Coronary Artery Disease: An Analysis of 136,905 Hospitalizations in Get With The Guidelines,” *American Heart Journal* 157, no. 1 (2009): 111 -7.e2, <https://doi.org/10.1016/j.ahj.2008.08.010>.
- 25 Olga Castañer et al., “Remnant Cholesterol, Not LDL Cholesterol, Is Associated with Incident Cardiovascular Disease,” *Journal of the American College of Cardiology* 76, no. 23 (2020): 2712–24, <https://doi.org/10.1016/j.jacc.2020.10.008>.

- 26 Nicholas G. Norwitz et al., “Elevated LDL Cholesterol with a Carbohydrate-Restricted Diet: Evidence for a ‘Lean Mass Hyper-Responder’ Phenotype,” *Current Developments in Nutrition* 6, no. 1 (November 2021): nzab144, <https://doi.org/10.1093/cdn/nzab144>.
- 27 Christopher N. Blesso and Maria Luz Fernandez, “Dietary Cholesterol, Serum Lipids, and Heart Disease: Are Eggs Working for or Against You?,” *Nutrients* vol. 10, no. 4 (2018): 426, doi:10.3390/nu10040426.
- 28 Fred Kern Jr., “Normal Plasma Cholesterol in an 88-Year-Old Man Who Eats 25 Eggs a Day: Mechanisms of Adaptation,” *New England Journal of Medicine* 324 (1991): 896–9, <https://doi.org/10.1056/NEJM199103283241306>.
- 29 Yee-Wen Huang et al., “Vegan Diet and Blood Lipid Profiles: A Cross-Sectional Study of Pre and Postmenopausal Women,” *BMC Women’s Health* 14 (2014): 55, <https://doi.org/10.1186/1472-6874-14-55>.
- 30 B. Duggan and H. O’Kane, “Hypercholesterolaemia in a Vegan,” *The Ulster Medical Journal* 66, no. 1 (1997): 57–8.
- 31 Amelie Scheu et al., “The Genetic Prehistory of Domesticated Cattle from Their Origin to the Spread Across Europe,” *BMC Genetics* 16, no. 54 (2015), <https://doi.org/10.1186/s12863-015-0203-2>.
- 32 Meilan Solly, “Prehistoric Farmers’ Teeth Show Humans Were Drinking Animal Milk 6,000 Years Ago,” *Smithsonian Magazine*, September 11, 2019, <https://www.smithsonianmag.com/smart-news/prehistoric-farmers-teeth-show-humans-were-drinking-animal-milk-6000-years-ago-180973101/>.
- 33 Debashree Roy, Aiqian Ye, Paul J. Moughan, and Harjinder Singh, “Composition, Structure, and Digestive Dynamics of Milk from Different Species: A Review,” *Frontiers in Nutrition* 7 (October 2020): 577759, <https://doi.org/10.3389/fnut.2020.577759>.
- 34 Gitanjali M. Singh et al., “Global, Regional, and National Consumption of Sugar-Sweetened Beverages, Fruit Juices, and Milk: A Systematic Assessment of Beverage Intake in 187 Countries,” *PLoS ONE* 10, no. 8 (2015): e0124845, <https://doi.org/10.1371/journal.pone.0124845>.
- 35 Suvi T. Itkonen, Maijaliisa Erkkola, and Christel J. E. Lamberg-Allardt, “Vitamin D Fortification of Fluid Milk Products and Their Contribution to Vitamin D Intake and Vitamin D Status in Observational Studies—A Review” *Nutrients* 10, no. 8 (2018): 1054,

- <https://doi.org/10.3390/nu10081054>.
- 36 Olivia L. van der Reijden, Michael B. Zimmermann, and Valeria Galetti, “Iodine in Dairy Milk: Sources, Concentrations and Importance to Human Health,” *Best Practice & Research: Clinical Endocrinology & Metabolism* 31, no. 4 (2017): 385–95, <https://doi.org/10.1016/j.beem.2017.10.004>.
- 37 W. P. Weiss, J. M. Pinos-Rodríguez, and M. T. Socha, “Effects of Feeding Supplemental Organic Iron to Late Gestation and Early Lactation Dairy Cows,” *Journal of Dairy Science* 93, no. 5 (May 2010): 2153–60, <https://doi.org/10.3168/jds.2010-3051>.
- 38 Charles M. Benbrook et al., “Enhancing the Fatty Acid Profile of Milk Through Forage-Based Rations, with Nutrition Modeling of Diet Outcomes,” *Food Science & Nutrition* 6, no. 3 (2018):681–700, <https://doi.org/10.1002/fsn3.610>.
- 39 R. Ranjan, A. Ranjan, G. S. Dhaliwal, and R. C. Patra, “L-Ascorbic Acid (Vitamin C) Supplementation to Optimize Health and Reproduction in Cattle,” *The Veterinary Quarterly* 32, no. 3–4 (2012): 145–50, <https://doi.org/10.1080/01652176.2012.734640>.
- 40 Andrea S. Wiley, “Lactose Intolerance.” *Evolution, Medicine, and Public Health* 2020, no. 1 (February 2020): 47–8, <https://doi.org/10.1093/emph/eoaa006>.
- 41 Christian Løvold Storhaug et al., “Country, Regional, and Global Estimates for Lactose Malabsorption in Adults: A Systematic Review and Meta-Analysis,” *The Lancet: Gastroenterology & Hepatology* 2, no. 10 (2017): 738–46, [https://doi.org/10.1016/S2468-1253\(17\)30154-1](https://doi.org/10.1016/S2468-1253(17)30154-1).
- 42 Michael de Vrese et al., “Probiotics—Compensation for Lactase Insufficiency,” *The American Journal of Clinical Nutrition* 73, no. 2 (February 2001): 421s–9s, <https://doi.org/10.1093/ajcn/73.2.421s>.
- 43 Timothy J. Wilt et al., *Lactose Intolerance and Health*, Evidence Report/Technology Assessment No. 192, Agency for Healthcare Research and Quality Publication No. 10-E004. (Rockville, MD: Agency for Healthcare Research and Quality, 2010).
- 44 Clair-Yves Boquien, “Human Milk: An Ideal Food for Nutrition of Preterm Newborn,” *Frontiers in Pediatrics* 6 (2018): 295, <https://doi.org/10.3389/fped.2018.00295>.
- 45 Keith Bernard Woodford, “Casomorphins and Gliadorphins Have

Diverse Systemic Effects Spanning Gut, Brain and Internal Organs,” *International Journal of Environmental Research and Public Health* 18, no. 15 (2021): 7911, <https://doi.org/10.3390/ijerph18157911>.

- [46](#) Simon Brooke-Taylor, Karen Dwyer, Keith Woodford, and Natalya Kost, “Systematic Review of the Gastrointestinal Effects of A1 Compared with A2 β -Casein,” *Advances in Nutrition* 8, no. 5 (2017): 739–48, <https://doi.org/10.3945/an.116.013953>.
- [47](#) <https://pubmed.ncbi.nlm.nih.gov/28790893/>.
- [48](#) Raffaele Falsaperla et al., “Epileptic Seizures as a Manifestation of Cow’s Milk Allergy: A Studied Relationship and Description of Our Pediatric Experience.” *Expert Review of Clinical Immunology* 10, no. 12 (2014): 1597–609, <https://doi.org/10.1586/1744666X.2014.977259>; Raffaele Falsaperla et al., The Gut-Brain Axis: A New Pathogenic View of Neurologic Symptoms—Description of a Pediatric Case. *Journal of Pediatric Neurosciences* 12, no. 1 (2017): 105–8, https://doi.org/10.4103/jpn.JPN_190_16.
- [49](#) Pablo José González-Domenech et al., “A Narrative Review about Autism Spectrum Disorders and Exclusion of Gluten and Casein from the Diet,” *Nutrients* 14, no. 9 (2022): 1797, <https://doi.org/10.3390/nu14091797>.
- [50](#) C. Hoppe et al., “Differential Effects of Casein Versus Whey on Fasting Plasma Levels of Insulin, IGF-1 and IGF-1/IGFBP-3: Results from a Randomized 7-Day Supplementation Study in Prepubertal Boys.” *European Journal of Clinical Nutrition* 63, no. 9 (2009): 1076–83, <https://doi.org/10.1038/ejcn.2009.34>.
- [51](#) Bodo C. Melnik, “Lifetime Impact of Cow’s Milk on Overactivation of mTORC1: From Fetal to Childhood Overgrowth, Acne, Diabetes, Cancers, and Neurodegeneration,” *Biomolecules* 11, no. 3 (2021): 404, <https://doi.org/10.3390/biom11030404>.
- [52](#) Hao Hong et al., “Central IGF1 Improves Glucose Tolerance and Insulin Sensitivity in Mice.” *Nutrition & Diabetes* 7, no. 12 (2017): 2, <https://doi.org/10.1038/s41387-017-0002-0>.
- [53](#) E. Sienkiewicz-Szlapka, “Contents of Agonistic and Antagonistic Opioid Peptides in Different Cheese Varieties,” *International Dairy Journal* 19, no. 4 (2009): 258–63, <https://doi.org/10.1016/j.idairyj.2008.10.011>.

- 54 Rachel L. Adams and Kenneth Shane Broughton, “Insulinotropic Effects of Whey: Mechanisms of Action, Recent Clinical Trials, and Clinical Applications,” *Annals of Nutrition & Metabolism* 69, no. 1 (2016): 56–63, <https://doi.org/10.1159/000448665>.
- 55 C. Hoppe, “High Intakes of Milk, But Not Meat, Increase S-Insulin and Insulin Resistance in 8-Year-Old Boys,” *European Journal of Clinical Nutrition* 59 (2005): 393–8. <https://doi.org/10.1038/sj.ejcn.1602086>.
- 56 Walter C. Willett and David S. Ludwig, “Milk and Health,” *New England Journal of Medicine* 382, no. 7 (2020): 644–54, <https://doi.org/10.1056/NEJMra1903547>.
- 57 Ian J. Wallace, Clinton T. Rubin, and Daniel E. Lieberman, “Osteoporosis,” *Evolution, Medicine, and Public Health* 2015, no. 1 (2015): 343, <https://doi.org/10.1093/emph/eov032>.
- 58 J. J. Cao, “High Dietary Protein Intake and Protein-Related Acid Load on Bone Health,” *Current Osteoporosis Reports* 15, no. 6 (2017): 571–6, <https://doi.org/10.1007/s11914-017-0408-6>.

Chapter 12

- [1](#) J. Salas-Salvadó, P. Casas-Agustench, and A. Salas-Huetos, “Cultural and Historical Aspects of Mediterranean Nuts with Emphasis on Their Attributed Healthy and Nutritional Properties,” *Nutrition, Metabolism, and Cardiovascular Diseases: NMCD* 21, Suppl 1 (2011): S1–6, <https://doi.org/10.1016/j.numecd.2010.10.013>.
- [2](#) Michael Balter, “Ancient Waves of (Wild) Grain,” *Science*, June 22, 2009, <https://www.science.org/content/article/ancient-waves-wild-grain>.
- [3](#) Valentina Caracuta et al., “The Onset of Faba Bean Farming in the Southern Levant,” *Scientific Reports* 5 (2015): 14370, <https://doi.org/10.1038/srep14370>.
- [4](#) Marta Liber, Isabel Duarte, Ana Teresa Maia, and Hugo R. Oliveira, “The History of Lentil (*Lens culinaris* subsp. *culinaris*) Domestication and Spread as Revealed by Genotyping-by-Sequencing of Wild and Landrace Accessions,” *Frontiers in Plant Science* 12, (2021): 628439, <https://doi.org/10.3389/fpls.2021.628439>.
- [5](#) Yuval Noah Harari, *Sapiens: A Brief History of Humankind* (London: Vintage, 2011), 93-4.
- [6](#) A. H. Goodman, G. J. Armelagos, and J. C. Rose, “The Chronological Distribution of Enamel Hypoplasias from Prehistoric Dickson Mounds Populations,” *American Journal of Physical Anthropology* 65, no. 3 (1984): 259–66, <https://doi.org/10.1002/ajpa.1330650305>; Amanda Mummert, Emily Esche, Joshua Robinson, and George J. Armelagos, “Stature and Robusticity During the Agricultural Transition: Evidence from the Bioarchaeological Record,” *Economics and Human Biology* 9, no. 3 (2011): 284–301, <https://doi.org/10.1016/j.ehb.2011.03.004>.
- [7](#) Steven R. Hertzler, Jacqueline C. Lieblein-Boff, Mary Weiler, and Courtney Allgeier, “Plant Proteins: Assessing Their Nutritional Quality and Effects on Health and Physical Function,” *Nutrients* 12, no. 12 (2020): 3704, <https://doi.org/10.3390/nu12123704>.
- [8](#) Sara Avilés-Gaxiola, Cristina Chuck-Hernández, and Sergio O. Serna Saldívar, “Inactivation Methods of Trypsin Inhibitor in Legumes: A Review,” *Journal of Food Science* 83, no. 1 (2018):17–29, <https://doi.org/10.1111/1750-3841.13985>.

- 9 Seitan and tofu score are from Yohan Reynaud et al., “True Ileal Amino Acid Digestibility and Digestible Indispensable Amino Acid Scores (DIAASs) of Plant-Based Protein Foods,” *Food Chemistry* 338 (2021): 128020, <https://doi.org/10.1016/j.foodchem.2020.128020>; beef is from P. Ertl, W. Knaus, and W. Zollitsch, “An Approach to Including Protein Quality When Assessing the Net Contribution of Livestock to Human Food Supply,” *Animal: An International Journal of Animal Bioscience* 10, no. 11 (2016): 1883–9, <https://doi.org/10.1017/S175173116000902>; tilapia is from Nazma Shaheen et al., “Amino Acid Profiles and Digestible Indispensable Amino Acid Scores of Proteins from the Prioritized Key Foods in Bangladesh,” *Food Chemistry* 213 (2016): 83–9, <https://doi.org/10.1016/j.foodchem.2016.06.057>; all other values from Stuart M. Phillips, “Current Concepts and Unresolved Questions in Dietary Protein Requirements and Supplements in Adults,” *Frontiers in Nutrition* 4, (2017): 13, <https://doi.org/10.3389/fnut.2017.00013>.
- 10 Anthony F. Domenichiello, Alex P. Kitson, and Richard P. Bazinet, “Is Docosahexaenoic Acid Synthesis From α-Linolenic Acid Sufficient to Supply the Adult Brain?,” *Progress in Lipid Research* 59 (2015): 54–66, <https://doi.org/10.1016/j.plipres.2015.04.002>.
- 11 Burdge and Woottton, “Conversion of Alpha-Linolenic Acid.”
- 12 Lorenzo Anez-Bustillos et al., “Redefining Essential Fatty Acids in the Era of Novel Intravenous Lipid Emulsions.” *Clinical Nutrition* 37, no. 3 (2018): 784–9, <https://doi.org/10.1016/j.clnu.2017.07.004>.
- 13 Dinakaran Elango et al., “Raffinose Family Oligosaccharides: Friend or Foe for Human and Plant Health?,” *Frontiers in Plant Science* 13 (2022): 829118, <https://doi.org/10.3389/fpls.2022.829118>.
- 14 Fernando Fernández-Bañares, “Carbohydrate Malabsorption and Intolerance,” *Nutrients* 14, no. 9 (2022): 1923, <https://doi.org/10.3390/nu14091923>.
- 15 Dinakaran Elango et al., “Raffinose Family Oligosaccharides: Friend or Foe for Human and Plant Health?,” *Frontiers in Plant Science* 13 (2022): 829118, <https://doi.org/10.3389/fpls.2022.829118>.
- 16 Mrinal Samtiya, Rotimi E. Aluko, and Tejpal Dhewa, “Plant Food Anti-Nutritional Factors and Their Reduction Strategies: An Overview,” *Food Production, Processing and Nutrition* 2, no. 6 (2020), <https://doi.org/10.1186/s43014-020-0020-5>.

- 17 N. W. Solomons, R. A. Jacob, O. Pineda, and F. Viteri, “Studies on the Bioavailability of Zinc in Man. II. Absorption of Zinc From Organic and Inorganic Sources,” *The Journal of Laboratory and Clinical Medicine* 94, no. 2 (1979): 335–43.
- 18 Leif Hallberg, “Bioavailability of Dietary Iron in Man,” *Annual Review of Nutrition* 1 (1981): 123–47,
<https://doi.org/10.1146/annurev.nu.01.070181.001011>.
- 19 Mrinal Samtiya, Rotimi E. Aluko, and Tejpal Dhewa, “Plant Food Anti-Nutritional Factors and Their Reduction Strategies: An Overview,” *Food Production, Processing and Nutrition* 2, no. 6 (2020),
<https://doi.org/10.1186/s43014-020-0020-5>.
- 20 Juan Bernal, “Thyroid Hormones in Brain Development and Function,” in *Endotext*, ed. Kenneth R. Feingold et. al. (South Dartmouth (MA): MDText.com, Inc., 2000.), updated January 14, 2022,
<https://www.ncbi.nlm.nih.gov/books/NBK285549/>.
- 21 Daniel R. Doerge and Daniel M. Sheehan, “Goitrogenic and Estrogenic Activity of Soy Isoflavones,” *Environmental Health Perspectives* 110, Suppl. 3 (2002): 349–53, <https://doi.org/10.1289/ehp.02110s3349>.
- 22 Thozhukat Sathyapalan et al., “The Effect of Soy Phytoestrogen Supplementation on Thyroid Status and Cardiovascular Risk Markers in Patients with Subclinical Hypothyroidism: A Randomized, Double-Blind, Crossover Study,” *The Journal of Clinical Endocrinology and Metabolism* 96, no. 5 (2011): 1442–9, <https://doi.org/10.1210/jc.2010-2255>.
- 23 S. Hüser et al., “Effects of Isoflavones on Breast Tissue and the Thyroid Hormone System in humans: A Comprehensive Safety Evaluation,” *Archives of Toxicology* 92, no. 9 (2018): 2703–48,
<https://doi.org/10.1007/s00204-018-2279-8>.
- 24 Abdelsalam Elnour, Leif Hambræus, Mohammed Eltom, Michèle Dramaix, and Pierre Bourdoux, “Endemic Goiter with Iodine Sufficiency: A Possible Role for the Consumption of Pearl Millet in the Etiology of Endemic Goiter,” *The American Journal of Clinical Nutrition* 71, no. 1 (2000): 59–66, <https://doi.org/10.1093/ajcn/71.1.59>.
- 25 Han Wang et al., “Effect of Different Processing Methods on the Millet Polyphenols and Their Anti-Diabetic Potential,” *Frontiers in Nutrition* 9 (2022): 780499, <https://doi.org/10.3389/fnut.2022.780499>.

- 26 Anežka Adamcová, Kristian Holst Laursen, and Nicolai Zederkopff Ballin, “Lectin Activity in Commonly Consumed Plant-Based Foods: Calling for Method Harmonization and Risk Assessment,” *Foods (Basel, Switzerland)* 10, no. 11 (2021): 2796, <https://doi.org/10.3390/foods10112796>.
- 27 Abtar Mishra et al., “Structure-Function and Application of Plant Lectins in Disease Biology and Immunity,” *Food and Chemical Toxicology: An International Journal Published for the British Industrial Biological Research Association* 134 (2019): 110827, <https://doi.org/10.1016/j.fct.2019.110827>.
- 28 M. Lopez-Moreno and M. Miguel, “Antinutrients: Lectins, Goitrogens, Phytates and Oxalates, Friends or Foe?,” *Journal of Functional Foods* 89 (2022): 104938, <https://doi.org/10.1016/j.jff.2022.104938>.
- 29 Alessio Fasano, “All Disease Begins in the (Leaky) Gut: Role of Zonulin-Mediated Gut Permeability in the Pathogenesis of Some Chronic Inflammatory Diseases,” *F1000Research* 9 (2020): F1000 Faculty Rev-69, <https://doi.org/10.12688/f1000research.20510.1>.
- 30 Michael Camilleri, “Leaky Gut: Mechanisms, Measurement and Clinical Implications in Humans,” *Gut* 68, no. 8 (2019): 1516–26, <https://doi.org/10.1136/gutjnl-2019-318427>.
- 31 Qiang Wang et al., “Identification of Intact Peanut Lectin in Peripheral Venous Blood,” *Lancet (London, England)* 352, no. 9143 (1998): 1831–2, [https://doi.org/10.1016/S0140-6736\(05\)79894-9](https://doi.org/10.1016/S0140-6736(05)79894-9).
- 32 Aristo Vojdani, Daniel Afar, and Elroy Vojdani, “Reaction of Lectin-Specific Antibody with Human Tissue: Possible Contributions to Autoimmunity,” *Journal of Immunology Research* 2020 (2020): 1438957, <https://doi.org/10.1155/2020/1438957>.
- 33 L. Anselmi et al., “Ingestion of Subthreshold Doses of Environmental Toxins Induces Ascending Parkinsonism in the Rat,” *NPJ Parkinson’s Disease* 4 (2018): 30, <https://doi.org/10.1038/s41531-018-0066-0>.
- 34 Anežka Adamcová, Kristian Holst Laursen, and Nicolai Zederkopff Ballin, “Lectin Activity in Commonly Consumed Plant-Based Foods: Calling for Method Harmonization and Risk Assessment,” *Foods (Basel, Switzerland)* 10, no. 11 (2021): 2796, <https://doi.org/10.3390/foods10112796>.
- 35 EFSA Panel on Contaminants in the Food Chain (CONTAM) et al.,

“Evaluation of the Health Risks Related to the Presence of Cyanogenic Glycosides in foods Other Than Raw Apricot Kernels.” *EFSA Journal: European Food Safety Authority* 17, no. 4 (2019): e05662, <https://doi.org/10.2903/j.efsa.2019.5662>.

- 36 Food Standards Australia New Zealand, “Survey of Cyanogenic Glycosides in Plant-Based Foods in Australia and New Zealand 2010–13,” FSANZ (April 2014): 1–78, <https://www.foodstandards.gov.au/consumer/chemicals/cassava/Documents/FINAL%20report%20on%20survey%20of%20cyanogenic%20glycosides%20in%20plant-based%20foods.pdf>.
- 37 Sergio Gutiérrez et al., “The Human Digestive Tract Has Proteases Capable of Gluten Hydrolysis,” *Molecular Metabolism* 6, no. 7 (2017): 693–702, <https://doi.org/10.1016/j.molmet.2017.05.008>.
- 38 Emma Clappison, Marios Hadjivassiliou, and Panagiotis Zis, “Psychiatric Manifestations of Coeliac Disease, a Systematic Review and Meta-Analysis,” *Nutrients* 12, no. 1 (2020): 142, <https://doi.org/10.3390/nu12010142>.
- 39 Marco A. Paez et al., “Delay in Diagnosis of Celiac Disease in Patients Without Gastrointestinal Complaints,” *The American Journal of Medicine* 130, no. 11 (2017): 1318–23, <https://doi.org/10.1016/j.amjmed.2017.05.027>.
- 40 Leszek Rudzki and Agata Szulc, “‘Immune Gate’ of Psychopathology: The Role of Gut Derived Immune Activation in Major Psychiatric Disorders,” *Frontiers in Psychiatry* 9 (2018): 205, <https://doi.org/10.3389/fpsyg.2018.00205>.
- 41 Emily G. Severance et al., “IgG Dynamics of Dietary Antigens Point to Cerebrospinal Fluid Barrier or Flow Dysfunction in First-Episode Schizophrenia,” *Brain, Behavior, and Immunity* 44 (2015): 148–58, <https://doi.org/10.1016/j.bbi.2014.09.009>.
- 42 Paola Bressan and Peter Kramer, “Bread and Other Edible Agents of Mental Disease,” *Frontiers in Human Neuroscience* 10 (2016): 130, <https://doi.org/10.3389/fnhum.2016.00130>.
- 43 Eleanor Busby et al., “Mood Disorders and Gluten: It’s Not All in Your Mind! A Systematic Review with Meta-Analysis,” *Nutrients* 10, no. 11 (2018): 1708, <https://doi.org/10.3390/nu10111708>.
- 44 Elena Lionetti et al., “Gluten Psychosis: Confirmation of a New Clinical

- Entity,” *Nutrients* 7, no. 7 (2015): 5532–9,
<https://doi.org/10.3390/nu7075235>.
- 45 Carmen Costas-Ferreira, Rafael Durán, and Lilian R. F. Faro, “Toxic Effects of Glyphosate on the Nervous System: A Systematic Review,” *International Journal of Molecular Sciences* 23, no. 9 (2022): 4605,
<https://doi.org/10.3390/ijms23094605>.
- 46 Maria Gloria Mumolo et al., “Is Gluten the Only Culprit for Non-Celiac Gluten/Wheat Sensitivity?,” *Nutrients* 12, no. 12 (2020): 3785,
<https://doi.org/10.3390/nu12123785>.
- 47 US Department of Agriculture and US Department of Health and Human Services. *Dietary Guidelines for Americans, 2020–2025*. 9th Edition. (December 2020): 20, 146. Available at DietaryGuidelines.gov.
- 48 “Starchy Foods and Carbohydrates,” NHS, last updated February 26, 2020, <https://www.nhs.uk/live-well/eat-well/food-types/starchy-foods-and-carbohydrates/>.
- 49 Andrew Reynolds et al., “Carbohydrate Quality and Human Health: A Series of Systematic Reviews and Meta-Analyses,” *Lancet (London, England)* 393, no. 10170 (2019): 434–45,
[https://doi.org/10.1016/S0140-6736\(18\)31809-9](https://doi.org/10.1016/S0140-6736(18)31809-9).
- 50 U.S. Department of Agriculture and U.S. Department of Health and Human Services. *Dietary Guidelines for Americans, 2020–2025*. 9th Edition. (December 2020): ix. Available at DietaryGuidelines.gov.
- 51 “Staple Foods: What Do People Eat?,” Food and Agricultural Organization of the United Nations, accessed June 5, 2023,
<https://www.fao.org/3/u8480e/u8480e07.htm>.

Chapter 13

- [1](#) U.S. Department of Agriculture and U.S. Department of Health and Human Services, *Dietary Guidelines for Americans, 2020–2025*. 9th ed. (December 2020), <https://www.DietaryGuidelines.gov>.
- [2](#) Martin L. Cipollini and Douglas J. Levey, “Secondary Metabolites of Fleshy Vertebrate-Dispersed Fruits: Adaptive Hypotheses and Implications for Seed Dispersal,” *The American Naturalist* 150, no. 3 (1997): 346–72, <https://doi.org/10.1086/286069>.
- [3](#) N. H. Strickland, “Eating a Manchineel ‘Beach Apple’,” *BMJ (Clinical research ed.)* 321, no. 7258 (2000): 428, <https://doi.org/10.1136/bmj.321.7258.428>.
- [4](#) Eduardo H. Rapoport and Barbara S. Drausal, “Edible Plants,” in *Encyclopedia of Biodiversity*, ed. Simon A. Levin, 2nd ed., 3 (Elsevier, 2013), 127–32, <http://dx.doi.org/10.1016/B978-0-12-384719-5.00160-X>.
- [5](#) Julie Dunne et al., “Earliest Direct Evidence of Plant Processing in Prehistoric Saharan Pottery,” *Nature Plants* 3 (2016): 16194, <https://doi.org/10.1038/nplants.2016.194>.
- [6](#) Rapoport and Drausal, “Edible Plants.”
- [7](#) W.P.T. James et al., “Nutrition and Its Role in Human Evolution,” *Journal of Internal Medicine*, 285 (2019): 534, <https://doi.org/10.1111/joim.12878>.
- [8](#) Bressan and Kramer, “Bread and Other Edible Agents,” 2.
- [9](#) Adam Drewnowski, “Concept of a Nutritious Food: Toward a Nutrient Density Score,” *The American Journal of Clinical Nutrition* 82, no. 4 (2005): 721–32, <https://doi.org/10.1093/ajcn/82.4.721>.
- [10](#) Rebecca M. Lovell and Alexander C. Ford, “Global Prevalence of and Risk Factors for Irritable Bowel Syndrome: A Meta-Analysis,” *Clinical Gastroenterology and Hepatology* 10, no. 7 (2012): 712–21.e4, <https://doi.org/10.1016/j.cgh.2012.02.029>.
- [11](#) Mohammad Zamani, Shaghayegh Alizadeh-Tabari, and Vahid Zamani, “Systematic Review with Meta-Analysis: The Prevalence of Anxiety and Depression in Patients with Irritable Bowel Syndrome,” *Alimentary Pharmacology & Therapeutics* 50, no. 2 (2019): 132–43, <https://doi.org/10.1111/apt.15325>.

- 12 Hannah Mitchell et al., “Review Article: Implementation of a Diet Low in FODMAPs for Patients with Irritable Bowel Syndrome: Directions for Future Research,” *Alimentary Pharmacology & Therapeutics* 49, no. 2 (2019): 124–39, <https://doi.org/10.1111/apt.15079>.
- 13 Amy Fedewa and Satish S. C. Rao, “Dietary Fructose Intolerance, Fructan Intolerance and FODMAPs,” *Current Gastroenterology Reports* 16, no. 1 (2014): 370, <https://doi.org/10.1007/s11894-013-0370-0>.
- 14 Nancy D. Turner and Joanne R. Lupton, “Dietary Fiber,” *Advances in Nutrition* 2, no. 2 (2011): 151–2, <https://doi.org/10.3945/an.110.000281>.
- 15 Kok-Yang Tan and Francis Seow-Choen, “Fiber and Colorectal Diseases: Separating Fact from Fiction,” *World Journal of Gastroenterology* 13, no. 31 (2007): 4161–7, <https://doi.org/10.3748/wjg.v13.i31.4161>.
- 16 Ruben D. Acosta and Brooks D. Cash, “Clinical Effects of Colonic Cleansing for General Health Promotion: A Systematic Review,” *The American Journal of Gastroenterology* 104, no. 11 (2009): 2830–6; quiz 2837, <https://doi.org/10.1038/ajg.2009.494>.
- 17 William D. Rees et al., “Regenerative Intestinal Stem Cells Induced by Acute and Chronic Injury: The Saving Grace of the Epithelium?,” *Frontiers in Cell and Developmental Biology* 8 (2020): 583919, <https://doi.org/10.3389/fcell.2020.583919>.
- 18 Johnson W. McRorie, Jr. and Nicola M. McKeown, “Understanding the Physics of Functional Fibers in the Gastrointestinal Tract: An Evidence-Based Approach to Resolving Enduring Misconceptions about Insoluble and Soluble Fiber,” *Journal of the Academy of Nutrition and Dietetics* 117, no. 2 (2017): 251–64, <https://doi.org/10.1016/j.jand.2016.09.021>.
- 19 Ghada A. Soliman, “Dietary Fiber, Atherosclerosis, and Cardiovascular Disease,” *Nutrients* 11, no. 5 (2019): 1155, <https://doi.org/10.3390/nu11051155>.
- 20 Kathleen J. Melanson et al., “Consumption of Whole-Grain Cereals during Weight Loss: Effects on Dietary Quality, Dietary Fiber, Magnesium, Vitamin B-6, and Obesity,” *Journal of the American Dietetic Association* 106, no. 9 (2006): 1380–8; quiz 1389–90, <https://doi.org/10.1016/j.jada.2006.06.003>.
- 21 Karen D. Corbin et al., “Host-Diet-Gut Microbiome Interactions Influence Human Energy Balance: A Randomized Clinical Trial,”

- Nature Communications* 14, no. 1 (2023): 3161,
<https://doi.org/10.1038/s41467-023-38778-x>.
- 22 Ron Sender, Shai Fuchs, and Ron Milo, “Are We Really Vastly Outnumbered? Revisiting the Ratio of Bacterial to Host Cells in Humans,” *Cell* 164, no. 3 (2016): 337–40,
<https://doi.org/10.1016/j.cell.2016.01.013>.
- 23 Peixin Fan et al., “Metabolites of Dietary Protein and Peptides by Intestinal Microbes and their Impacts on Gut,” *Current Protein & Peptide Science* 16, no. 7 (2015): 646–54,
<https://doi.org/10.2174/138920371666150630133657>.
- 24 Yun-Hee Youm et al., “The Ketone Metabolite β -Hydroxybutyrate Blocks NLRP3 Inflammasome-Mediated Inflammatory Disease,” *Nature Medicine* 21, no. 3 (2015): 263–9,
<https://doi.org/10.1038/nm.3804>.
- 25 Lawrence A. David et al., “Diet Rapidly and Reproducibly Alters the Human Gut Microbiome,” *Nature* 505, no. 7484 (2014): 559–63,
<https://doi.org/10.1038/nature12820>.
- 26 J. Horn, D. E. Mayer, S. Chen, and E. A. Mayer, “Role of Diet and Its Effects on the Gut Microbiome in the Pathophysiology of Mental Disorders,” *Translational Psychiatry* 12, no. 1 (2022): 164,
<https://doi.org/10.1038/s41398-022-01922-0/>
- 27 EFSA Panel on Contaminants in the Food Chain (CONTAM) et al., “Risk Assessment of Glycoalkaloids in Feed and Food, in Particular in Potatoes and Potato-Derived Products,” *EFSA Journal* 18, no. 8 (2020): e06222, <https://doi.org/10.2903/j.efsa.2020.6222>.
- 28 Mahmoud Bagheri, Ali Akbar Shahnejat Bushehri, Mohammad Reza Hassandokht, and Mohammad Reza Naghavi, “Evaluation of Solasonine Content and Expression Patterns of *SGT1* Gene in Different Tissues of Two Iranian Eggplant (*Solanum melongena* L.) Genotypes,” *Food Technology and Biotechnology* 55, no. 2 (2017): 236–42,
<https://doi.org/10.17113/ftb.55.02.17.4883>.
- 29 Mendel Friedman, “Chemistry and Anticarcinogenic Mechanisms of Glycoalkaloids Produced by Eggplants, Potatoes, and Tomatoes,” *Journal of Agricultural and Food Chemistry* 63, no. 13 (2015): 3323–37, <https://doi.org/10.1021/acs.jafc.5b00818>.
- 30 Michael Adams, Matthias Wiedenmann, Gerolf Tittel, and Rudolf Bauer,

“HPLC-MS Trace Analysis of Atropine in *Lycium barbarum* Berries,” *Phytochemical Analysis* 17, no. 5 (2006): 279–83, <https://doi.org/10.1002/pca.915>.

- 31 EFSA Panel on Contaminants in the Food Chain (CONTAM) et al., “Risk Assessment of Glycoalkaloids in Feed and Food, in Particular in Potatoes and Potato-Derived Products,” *EFSA Journal: European Food Safety Authority* 18, no. 8 (2020): e06222, <https://doi.org/10.2903/j.efsa.2020.6222>.
- 32 Victor Fattori et al., “Capsaicin: Current Understanding of Its Mechanisms and Therapy of Pain and Other Pre-Clinical and Clinical Uses,” *Molecules* 21, no. 7 (2016): 844, <https://doi.org/10.3390/molecules21070844>.
- 33 K. Czaja, G. A. Burns, and R. C. Ritter, “Capsaicin-Induced Neuronal Death and Proliferation of the Primary Sensory Neurons Located in the Nodose Ganglia of Adult Rats,” *Neuroscience* 154, no. 2 (2008): 621–30, <https://doi.org/10.1016/j.neuroscience.2008.03.055>.
- 34 Michał Pasierski and Bartłomiej Szulczyk, “Beneficial Effects of Capsaicin in Disorders of the Central Nervous System,” *Molecules* 27, no. 8 (2022): 2484, <https://doi.org/10.3390/molecules27082484>.
- 35 “Too Hot Isn’t Healthy—Foods with Very High Capsaicin Concentrations Can Damage Health” BfR Opinion No. 053/2011, Bundesinstitut für Risikobewertung, October 18, 2011, <https://www.bfr.bund.de/cm/349/too-hot-isnt-healthy-foods-with-very-high-capsaicin-concentrations-can-damage-health.pdf>.
- 36 Njoku Damian Ndubuisi and Ano Chukwuka Ugochukwu Chidiebere, “Cyanide in Cassava: A Review,” *International Journal of Genomics and Data Mining* 2, no. 1 (2018): 118, <https://doi.org/10.29011/2577-0616.000118>.
- 37 Ndubuisi and Chidiebere, “Cyanide in Cassava.”
- 38 Alicia A. Quinn, Harry Myrans, and Roslyn M. Gleadow, “Cyanide Content of Cassava Food Products Available in Australia,” *Foods* 11, no. 10 (2022): 1384, <https://doi.org/10.3390/foods11101384>.
- 39 Phoebe H. Alitubeera et al., “Outbreak of Cyanide Poisoning Caused by Consumption of Cassava Flour—Kasese District, Uganda, September 2017,” *Morbidity and Mortality Weekly Report* 68, no. 13 (2019): 308–11, <https://www.cdc.gov/mmwr/volumes/68/wr/mm6813a3.htm>.

- 40 Alitubeera et al., “Outbreak of Cyanide Poisoning,”
- 41 Espérance Kashala-Abotnes et al., “Konzo: A Distinct Neurological Disease Associated with Food (Cassava) Cyanogenic Poisoning,” *Brain Research Bulletin* 145 (2019): 87–91,
<https://doi.org/10.1016/j.brainresbull.2018.07.001>.
- 42 M. A. Prieto, Cecilia Jiménez López, and Jesus Simal-Gandara, “Glucosinolates: Molecular Structure, Breakdown, Genetic, Bioavailability, Properties and Healthy and Adverse Effects,” *Advances in Food and Nutrition Research* 90 (2019): 305–350,
<https://doi.org/10.1016/bs.afnr.2019.02.008>.
- 43 Prieto, López, and Simal-Gandara, “Glucosinolates.”
- 44 Peter Felker, Ronald Bunch, and Angela M. Leung, “Concentrations of Thiocyanate and Goitrin in Human Plasma, Their Precursor Concentrations in Brassica Vegetables, and Associated Potential Risk for Hypothyroidism,” *Nutrition Reviews* 74, no. 4 (2016): 248–58,
<https://doi.org/10.1093/nutrit/nuv110>.

Chapter 14

- [1](#) Deanna M. Minich, “A Review of the Science of Colorful, Plant-Based Food and Practical Strategies for ‘Eating the Rainbow,’” *Journal of Nutrition and Metabolism* 2019 (2019): 2125070, <https://doi.org/10.1155/2019/2125070>.
- [2](#) Jens Lykkesfeldt and Henrik E. Poulsen, “Is Vitamin C Supplementation Beneficial? Lessons Learned from Randomised Controlled Trials,” *British Journal of Nutrition* 103, no. 9 (2010): 1251–59, <https://doi.org/10.1017/S0007114509993229>.
- [3](#) Irina Robinson, Daniela Gonzalez de Serna, Absalon Gutierrez, and David S. Schade, “Vitamin E in Humans: An Explanation of Clinical Trial Failure,” *Endocrine Practice: Official Journal of the American College of Endocrinology and the American Association of Clinical Endocrinologists* 12, no. 5 (2006): 576–82, <https://doi.org/10.4158/EP.12.5.576>.
- [4](#) Homer S. Black, Fritz Boehm, Ruth Edge, and T. George Truscott, “The Benefits and Risks of Certain Dietary Carotenoids that Exhibit Both Anti- and Pro-Oxidative Mechanisms: A Comprehensive Review,” *Antioxidants (Basel, Switzerland)* 9, no. 3 (March 2020): 264, <https://doi.org/10.3390/antiox9030264>.
- [5](#) Deanna M. Minich, “A Review of the Science.”
- [6](#) Agricultural Research Service, “Oxygen Radical Absorbance Capacity (ORAC) of Selected Foods, Release 2 (2010),” United States Department of Agriculture, Updated August 13, 2016, Accessed January 9, 2023, <http://www.ars.usda.gov/Services/docs.htm?docid=15866>; Archived at <https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/nutrient-data-laboratory/docs/oxygen-radical-absorbance-capacity-orac-of-selected-foods-release-2-2010/>.
- [7](#) Alan Crozier, Indu B Jaganath, and Michael N Clifford, “Dietary Phenolics: Chemistry, Bioavailability and Effects on Health, *Natural Product Reports* 26, no. 8 (2009): 1001–43, <https://doi.org/10.1039/b802662ap> [quote is from page 1002].
- [8](#) Alan Crozier, Indu B Jaganath, and Michael N Clifford, “Dietary

- Phenolics.”
- 9 Alan Crozier, Indu B Jaganath, and Michael N Clifford, “Dietary Phenolics” [quote is from page 1039].
- 10 Doug Bierend, “How Blueberries Became a Superfood,” *Outside*, Apr 23, 2019, <https://www.outsideonline.com/health/nutrition/blueberries-superfood-benefits>.
- 11 Doug Bierend, “How Blueberries Became a Superfood.”
- 12 Doug Bierend, “How Blueberries Became a Superfood.”
- 13 Emma Weitkamp and Torill Eidsvaag, “Agenda Building In Media Coverage of Food Research,” *Journalism Practice* 8, no. 6 (2014): 871–86, <https://doi.org/10.1080/17512786.2013.865966>.
- 14 Phuong H. L. Tran and Thao T. D. Tran, “Blueberry Supplementation in Neuronal Health and Protective Technologies for Efficient Delivery of Blueberry Anthocyanins,” *Biomolecules* 11, no. 1 (2021): 102, <https://doi.org/10.3390/biom11010102>.
- 15 Nikolaj Travica, “The Effect of Blueberry Interventions on Cognitive Performance and Mood: A Systematic Review of Randomized Controlled Trials,” *Brain, Behavior, and Immunity* 85 (2020): 96–105, <https://doi.org/10.1016/j.bbi.2019.04.001>.
- 16 Joanna L. Bowtell et al., “Enhanced Task-Related Brain Activation and Resting Perfusion in Healthy Older Adults After Chronic Blueberry Supplementation,” *Applied Physiology, Nutrition, and Metabolism* 42, no. 7 (2017): 773–9, <https://doi.org/10.1139/apnm-2016-0550>.
- 17 Wolfgang Marx et al., “In Response to ‘There Is No Meta-Analytic Evidence of Blueberries Improving Cognitive Performance or Mood,’” *Brain, Behavior, and Immunity* 85 (2020): 193, <https://doi.org/10.1016/j.bbi.2019.10.001>.
- 18 US Department of Agriculture, “FoodData Central,” Agricultural Research Service, 2019, <https://fdc.nal.usda.gov>.
- 19 Marjorie L. McCullough et al., “Hypertension, the Kuna, and the Epidemiology of Flavanols,” *Journal of Cardiovascular Pharmacology* 47, Suppl 2 (2006): S103–9, <https://doi.org/10.1097/00005344-200606001-00003>.
- 20 Norman K. Hollenberg, “Vascular Action of Cocoa Flavanols in Humans: The Roots of the Story,” *Journal of Cardiovascular Pharmacology* 47, Suppl 2 (2006): S99–102,

- <https://doi.org/10.1097/00005344-200606001-00002>.
- 21 Hollenberg, “Vascular Action,” S101.
- 22 Corinna Zeli et al., “Chocolate and Cocoa-Derived Biomolecules for Brain Cognition during Ageing,” *Antioxidants* 11, no. 7 (2022): 1353, <https://doi.org/10.3390/antiox11071353>.
- 23 Iveta Bernatova, “Biological Activities of (-)-Epicatechin and (-)-Epicatechin-Containing Foods: Focus on Cardiovascular and Neuropsychological Health,” *Biotechnology Advances* 36, no. 3 (2018): 666–81, <https://doi.org/10.1016/j.biotechadv.2018.01.009>.
- 24 M. E. Alañón et al., “Assessment of Flavanol Stereoisomers and Caffeine and Theobromine Content in Commercial Chocolates,” *Food Chemistry* 208 (2016): 177–84, <https://doi.org/10.1016/j.foodchem.2016.03.116>.
- 25 Marielle Adrian and Philippe Jeandet, “Effects of Resveratrol on the Ultrastructure of Botrytis Cinerea Conidia and Biological Significance in Plant/Pathogen Interactions,” *Fitoterapia* 83, no. 8 (2012): 1345–50, <https://doi.org/10.1016/j.fitote.2012.04.004>.
- 26 John M. Pezzuto, “Resveratrol: Twenty Years of Growth, Development and Controversy,” *Biomolecules & Therapeutics* 27, no. 1 (2019): 1–14, <https://doi.org/10.4062/biomolther.2018.176>.
- 27 Alex J. T. Yang, Ahmed Bagit, and Rebecca E. K. MacPherson, “Resveratrol, Metabolic Dysregulation, and Alzheimer’s Disease: Considerations for Neurodegenerative Disease,” *International Journal of Molecular Sciences* 22, no. 9 (2021): 4628, <https://doi.org/10.3390/ijms22094628>.
- 28 Alex J. T. Yang, Ahmed Bagit, and Rebecca E. K. MacPherson, “Resveratrol, Metabolic Dysregulation, and Alzheimer’s Disease.”
- 29 Philippe Jeandet, Roger Bessis, Bernard F. Maume, and Mohamed Sbaghi, “Analysis of Resveratrol in Burgundy Wines,” *Journal of Wine Research* 4, no. 2 (1993): 79–85, <https://doi.org/10.1080/09571269308717954>.
- 30 José A. Hernández, Rosa C. López-Sánchez, and Adela Rendón-Ramírez, “Lipids and Oxidative Stress Associated with Ethanol-Induced Neurological Damage,” *Oxidative Medicine and Cellular Longevity* 2016 (2016): 1543809, <https://doi.org/10.1155/2016/1543809>.
- 31 E. Baraona and C. S. Lieber, “Effects of Ethanol on Lipid Metabolism,”

- Journal of Lipid Research* 20, no. 3 (1979): 289–315,
<https://doi.org/10.1016/j.jhep.2018.10.037>.
- 32 José A. Hernández, Rosa C. López-Sánchez, and Adela Rendón-Ramírez, “Lipids and Oxidative Stress Associated with Ethanol-Induced Neurological Damage,” *Oxidative Medicine and Cellular Longevity* 2016 (2016): 1543809, <https://doi.org/10.1155/2016/1543809>.
- 33 Teicholz, *The Big Fat Surprise*, 185–93.
- 34 W. C. Willett et al., “Mediterranean Diet Pyramid: A Cultural Model for Healthy Eating,” *The American Journal of Clinical Nutrition* 61, no. 6 Suppl (1995): 1402S–6S, <https://doi.org/10.1093/ajcn/61.6.1402S>.
- 35 Verena Jeschke, Jonathan Gershenson, and Daniel Giddings Vassão, “A Mode of Action of Glucosinolate-Derived Isothiocyanates: Detoxification Depletes Glutathione and Cysteine Levels with Ramifications on Protein Metabolism in *Spodoptera littoralis*.” *Insect Biochemistry and Molecular Biology* 71 (2016): 37–48, <https://doi.org/10.1016/j.ibmb.2016.02.002>.
- 36 Jed W. Fahey and Thomas W. Kensler, “The Challenges of Designing and Implementing Clinical Trials with Broccoli Sprouts... and Turning Evidence into Public Health Action,” *Frontiers in Nutrition* 8 (2021): 648788, <https://doi.org/10.3389/fnut.2021.648788>.
- 37 Fatemeh Ghazizadeh-Hashemi et al., “Efficacy and Safety of Sulforaphane for Treatment of Mild to Moderate Depression in Patients with History of Cardiac Interventions: A Randomized, Double-Blind, Placebo-Controlled Clinical Trial,” *Psychiatry and Clinical Neurosciences* 75, no. 8 (2021): 250–5, <https://doi.org/10.1111/pcn.13276>.
- 38 Greer McGuinness and Yeonsoo Kim, “Sulforaphane Treatment for Autism Spectrum Disorder: A Systematic Review,” *EXCLI Journal* 19 (2020): 892–903, <https://doi.org/10.17179/excli2020-2487>.
- 39 Valentina V. Huwiler et al., “Prolonged Isolated Soluble Dietary Fibre Supplementation in Overweight and Obese Patients: A Systematic Review with Meta-Analysis of Randomised Controlled Trials,” *Nutrients* 14, no. 13 (2022): 2627, <https://doi.org/10.3390/nu14132627>.
- 40 Industry Research, “Global Superfoods Market Size is Projected To Reach US\$ 287.75 Billion by 2027 | Superfoods Market Store, Delivery Options, Emerging Trends 2022 | Segmentation by Product Type,

Applications, Regions, & Key-Players (ADM, Ardent Mills, Bunge)," Globe Newswire, February 28, 2022,
<https://www.globenewswire.com/en/news-release/2022/02/28/2393441/0/en/Global-Superfoods-Market-Size-is-Projected-To-Reach-US-287-75-Billion-by-2027-Superfoods-Market-Store-Delivery-Options-Emerging-Trends-2022-Segmentation-by-Product-Type-Application.html>.

- ⁴¹ Georgia Ede, "The Antioxidant Myth," *Psychology Today*, December 30, 2017, <https://www.psychologytoday.com/us/blog/diagnosis-diet/201712/the-antioxidant-myth>.

Chapter 15

- [1](#) Ponmurugan Karuppiah, Muhammad Musthafa Poyil, Suresh S. S. Raja, and Imran Mohammad Mohammad, “Screening and Isolation of Vitamin B12 Producing *Pseudomonas* Sp. from Different Natural Sources,” *Annals of Phytomedicine* 10, no. 1 (2021): 249–54, <https://doi.org/10.21276/ap.2021.10.1.27>.
- [2](#) Alice H. Lichtenstein et al., “2021 Dietary Guidance to Improve Cardiovascular Health: A Scientific Statement from the American Heart Association,” *Circulation* 144, no. 23 (2021): e472–87, <https://doi.org/10.1161/CIR.0000000000001031>.
- [3](#) WHO European Office for the Prevention and Control of Noncommunicable Diseases, *Plant-Based Diets and Their Impact on Health, Sustainability and the Environment: A Review of the Evidence* (Copenhagen: WHO Regional Office for Europe, 2021), <https://apps.who.int/iris/bitstream/handle/10665/349086/WHO-EURO-2021-4007-43766-61591-eng.pdf>.
- [4](#) “What Does Plant-Based Mean to the Public?,” The Vegetarian Resource Group Blog, August 11, 2017, <https://www.vrg.org/blog/2017/08/11/what-does-plant-based-mean-to-the-public/>.
- [5](#) Eimear Leahy, Seán Lyonsa, and Richard S. J. Tol, “An Estimate of the Number of Vegetarians in the World,” ESRI Working Paper No. 340, March 2010, <https://www.esri.ie/system/files/media/file-uploads/2015-07/WP340.pdf>.
- [6](#) Georgios Paslakis et al., “Prevalence and Psychopathology of Vegetarians and Vegans: Results from a Representative Survey in Germany,” *Scientific Reports* 10, (2020): 6840, <https://doi.org/10.1038/s41598-020-63910-y>.
- [7](#) Paslakis et al., “Prevalence and Psychopathology”; Sarah Prescott Smith and Matthew Smith, “Meet Britain’s Vegans and Vegetarians,” YouGov, published January 20, 2022, <https://yougov.co.uk/topics/society/articles-reports/2022/01/20/meet-britains-vegans-and-vegetarians>.
- [8](#) Isabel Iguacel, Inge Huybrechts, Luis A. Moreno, and Nathalie Michels, “Vegetarianism and Veganism Compared with Mental Health and

Cognitive Outcomes: A Systematic Review and Meta-Analysis,” *Nutrition Reviews* 79, no. 4 (2021): 361–81, <https://doi.org/10.1093/nutrit/nuaa030>.

- 9 Dean Ornish et al., “Can Lifestyle Changes Reverse Coronary Heart Disease? The Lifestyle Heart Trial,” *Lancet (London, England)* 336, no. 8708 (1990): 129–33, [https://doi.org/10.1016/0140-6736\(90\)91656-u](https://doi.org/10.1016/0140-6736(90)91656-u).
- 10 Caldwell B. Esselstyn Jr., Stephen G. Ellis, Sharon V. Medendorp, and Timothy D. Crowe, “A Strategy to Arrest and Reverse Coronary Artery Disease: A 5-Year Longitudinal Study of a Single Physician’s Practice,” *The Journal of Family Practice* 41, no. 6 (1995): 560–8.
- 11 Matthew C. Riddle et al., “Consensus Report: Definition and Interpretation of Remission in Type 2 Diabetes,” *Diabetes Care*, 44, no. 10 (2021): 2438–44, <https://doi.org/10.2337/dc21-0034>.
- 12 Neal D. Barnard et al., “A Low-Fat Vegan Diet and a Conventional Diabetes Diet in the Treatment of Type 2 Diabetes: A Randomized, Controlled, 74-wk Clinical Trial,” *The American Journal of Clinical Nutrition* 89, no. 5 (2009): 1588S–96S, <https://doi.org/10.3945/ajcn.2009.26736H>.
- 13 Sarah J. Hallberg et al., “Effectiveness and Safety of a Novel Care Model for the Management of Type 2 Diabetes at 1 Year: An Open-Label, Non-Randomized, Controlled Study,” *Diabetes Therapy* 9, no. 2 (2018): 583–612, <https://doi.org/10.1007/s13300-018-0373-9>.
- 14 David Unwin et al., “What Predicts Drug-Free Type 2 Diabetes Remission? Insights from an 8-Year General Practice Service Evaluation of a Lower Carbohydrate Diet with Weight Loss,” *BMJ Nutrition, Prevention & Health* (2023): e000544, <https://doi.org/10.1136/bmjnph-2022-000544>.
- 15 Walter Willett et al., “Food in the Anthropocene: The EAT-Lancet Commission on Healthy Diets from Sustainable Food Systems,” *Lancet (London, England)* 393, no. 10170 (2019): 447–92, [https://doi.org/10.1016/S0140-6736\(18\)31788-4](https://doi.org/10.1016/S0140-6736(18)31788-4).
- 16 Willett et al., “Food in the Anthropocene,” 451–460.
- 17 Nicole Neufingerl and Ans Eilander, “Nutrient Intake and Status in Adults Consuming Plant-Based Diets Compared to Meat-Eaters: A Systematic Review,” *Nutrients* 14, no. 1 (2021): 29, <https://doi.org/10.3390/nu14010029>.

- 18 David O. Kennedy, “B Vitamins and the Brain: Mechanisms, Dose and Efficacy: A Review,” *Nutrients* 8, no. 2 (2016): 68, <https://doi.org/10.3390/nu8020068>.
- 19 Roser Granero et al., “The Role of Iron and Zinc in the Treatment of ADHD among Children and Adolescents: A Systematic Review of Randomized Clinical Trials,” *Nutrients* 13, no. 11 (2021):4059, <https://doi.org/10.3390/nu13114059>.
- 20 Herng-Sheng Lee et al., “Psychiatric Disorders Risk in Patients with Iron Deficiency Anemia and Association with Iron Supplementation Medications: A Nationwide Database Analysis,” *BMC Psychiatry* 20, no. 1 (2020): 216, <https://doi.org/10.1186/s12888-020-02621-0>.
- 21 Seyed Mojtaba Ghoreishy, Sara Ebrahimi Mousavi, Farzaneh Asoudeh, and Hamed Mohammadi, “Zinc Status in Attention-Deficit/Hyperactivity Disorder: A Systematic Review and Meta-Analysis of Observational Studies,” *Scientific Reports* 11, no. 1 (2021): 14612, <https://doi.org/10.1038/s41598-021-94124-5>; Granero et al., “The Role of Iron and Zinc.”
- 22 Matthew A. Petrilli et al., “The Emerging Role for Zinc in Depression and Psychosis,” *Frontiers in Pharmacology* 8 (2017): 414, <https://doi.org/10.3389/fphar.2017.00414>.
- 23 Petrilli et al., “The Emerging Role for Zinc.”
- 24 Elif Turan and Ozgul Karaaslan, “The Relationship between Iodine and Selenium Levels with Anxiety and Depression in Patients with Euthyroid Nodular Goiter,” *Oman Medical Journal* 35, no. 4 (2020): e161, <https://doi.org/10.5001/omj.2020.84>.
- 25 Klaus W. Lange, “Omega-3 Fatty Acids and Mental Health,” *Global Health Journal* 4, no. 1 (2020):18–30, <https://doi.org/10.1016/j.glohj.2020.01.004>.
- 26 Naveen Jayaram et al., “Vitamin B12 Levels and Psychiatric Symptomatology: A Case Series,” *The Journal of Neuropsychiatry and Clinical Neurosciences* 25, no. 2 (2013): 150–2, <https://doi.org/10.1176/appi.neuropsych.12060144>.
- 27 Aneel Kapoor et al., “Neuropsychiatric and Neurological Problems among Vitamin B12 Deficient Young Vegetarians,” *Neurosciences* 22, no. 3 (2017): 228–32, <https://doi.org/10.17712/nsj.2017.3.20160445>.
- 28 Akshata Huddar, Doniparthi Venkata Seshagiri, Subasree Ramakrishnan,

- and Raghavendra Kenchaiah, “Pearls & Oysters: Rapidly Reversible Dementia: Vitamin B12 Deficiency in a 29-Year-Old Woman,” *Neurology* 97, no. 6 (2021): e643–6, <https://doi.org/10.1212/WNL.00000000000012102>.
- 29 Laurie K. Mischley, “Conditionally Essential Nutrients: The State of the Science,” *Journal of Food and Nutrition* 1 (2014): 1–4, <http://www.jscholaronline.org/full-text/JFN/e204/Conditionally-Essential-Nutrients-The-State-of-the-Science.php>.
- 30 Harris Ripps and Wen Shen, “Review: Taurine: A ‘Very Essential’ Amino Acid,” *Molecular Vision* 18 (2012): 2673–86, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3501277>.
- 31 Karolina Karcz and Barbara Królak-Olejnik, “Vegan or Vegetarian Diet and Breast Milk Composition—A Systematic Review,” *Critical Reviews in Food Science and Nutrition* 61, no. 7 (2021): 1081–98, <https://doi.org/10.1080/10408398.2020.1753650>.
- 32 “Mise au Point de l’Académie royale de Médecine de Belgique sur le régime végétalien pour les enfants, femmes enceintes et allaitantes,” Académie Royale de Médecine de Belgique, May 14, 2019, https://www.armb.be/fileadmin/sites/armb/uploads/Document-site/pdf/Avis/2019/ARMB_Regime_vegetalien_.docx.pdf.
- 33 “Si la consommation d’un tel régime et ses conséquences sont de la responsabilité de l’adulte qui s’y soumet, il est tout à fait non recommandé médicalement et même proscrit de soumettre un enfant, en particulier lors des périodes de croissance rapides, à un régime potentiellement déstabilisant, justifiant des supplémentations et nécessitant des contrôles cliniques et biologiques fréquents. Ce concept d’alimentation où la supplémentation systématique et des contrôles sanguins obligatoires (accompagnement médical systématique par le généraliste et/ou le pédiatre) sont indispensables à l’exclusion de carences s’apparente non plus à une alimentation classique mais à une forme de « traitement » qu’il n’est pas éthique d’imposer à des enfants.” From Académie Royale de Médecine de Belgique, “Régimes végétariens et végétaliens administrés aux enfants et adolescents,” June 2019, 5–6, https://www.armb.be/fileadmin/sites/armb/uploads/Document-site/pdf/Avis/2019/ARMB_re_g_ve_ge_talien_version_complete.pdf

- 34 Carol L. Cheatham, “Nutritional Factors in Fetal and Infant Brain Development,” *Annals of Nutrition & Metabolism* 75, Suppl 1 (2019): 20–32, <https://doi.org/10.1159/000508052>.
- 35 Giorgia Sebastiani et al., “The Effects of Vegetarian and Vegan Diet during Pregnancy on the Health of Mothers and Offspring,” *Nutrients* 11, no. 3 (2019): 557, <https://doi.org/10.3390/nu11030557>.
- 36 Synne Groufh-Jacobsen et al., “Vegans, Vegetarians and Pescatarians Are at Risk of Iodine Deficiency in Norway,” *Nutrients* 12, no. 11 (2020): 3555, <https://doi.org/10.3390/nu12113555>; Cheatham, “Nutritional Factors in Fetal.”
- 37 Sanjay Basak, Rahul Mallick, and Asim K. Duttaroy, “Maternal Docosahexaenoic Acid Status during Pregnancy and Its Impact on Infant Neurodevelopment,” *Nutrients* 12, no. 12 (2020): 3615, <https://doi.org/10.3390/nu12123615>.
- 38 Graham C. Burdge, Sze-Yen Tan, and Christiani Jeyakumar Henry, “Long-Chain n-3 PUFA in Vegetarian Women: A Metabolic Perspective,” *Journal of Nutritional Science* 6 (2017): e58, <https://doi.org/10.1017/jns.2017.62>.
- 39 Robert K. McNamara, Jennifer J. Vannest, and Christina J. Valentine, “Role of Perinatal Long-Chain Omega-3 Fatty Acids in Cortical Circuit Maturation: Mechanisms and Implications for Psychopathology,” *World Journal of Psychiatry* 5, no. 1 (2015): 15–34, <https://doi.org/10.5498/wjp.v5.i1.15>.
- 40 Jan Krzysztof Blusztajn, Barbara E. Slack, and Tiffany J. Mellott, “Neuroprotective Actions of Dietary Choline,” *Nutrients* 9, no. 8 (2017): 815, <https://doi.org/10.3390/nu9080815>.
- 41 Jonathan G. Mun, LeeCole L. Legette, Chioma J. Ikonte, and Susan H. Mitmesser, “Choline and DHA in Maternal and Infant Nutrition: Synergistic Implications in Brain and Eye Health,” *Nutrients* 11, no. 5 (2019): 1125, <https://doi.org/10.3390/nu11051125>.
- 42 Scout McWilliams et al., “Iron Deficiency and Common Neurodevelopmental Disorders: A Scoping Review,” *PLoS ONE* 17, no. 9 (2022): e0273819, <https://doi.org/10.1371/journal.pone.0273819>.
- 43 Aline Marileen Wiegersma et al., “Association of Prenatal Maternal Anemia With Neurodevelopmental Disorders,” *JAMA Psychiatry* 76, no. 12 (2019): 1294–304,

- <https://doi.org/10.1001/jamapsychiatry.2019.2309>.
- 44 Sigrun Henjum et al., “Iron Status of Vegans, Vegetarians and Pescatarians in Norway,” *Biomolecules* 11, no. 3 (2021): 454, <https://doi.org/10.3390/biom11030454>.
- 45 Rakesh Balachandar, Raghu Pullakhandam, Bharati Kulkarni, and Harshpal Singh Sachdev, “Relative Efficacy of Vitamin D₂ and Vitamin D₃ in Improving Vitamin D Status: Systematic Review and Meta-Analysis,” *Nutrients* 13, no. 10 (2021): 3328, <https://doi.org/10.3390/nu13103328>.
- 46 Giorgia Sebastiani et al., “The Effects of Vegetarian and Vegan Diet during Pregnancy on the Health of Mothers and Offspring,” *Nutrients* 11, no. 3 (2019): 557, <https://doi.org/10.3390/nu11030557>.
- 47 Jing Wang et al., “Research Progress on the Role of Vitamin D in Autism Spectrum Disorder,” *Frontiers in Behavioral Neuroscience* 16 (2022): 859151, <https://doi.org/10.3389/fnbeh.2022.859151>.
- 48 Pinky Meena et al., “Sunlight Exposure and Vitamin D Status in Breastfed Infants,” *Indian Pediatrics* 54, no. 2 (2017): 105–111, <https://doi.org/10.1007/s13312-017-1010-9>.
- 49 Valentina Trimarco et al., “Insulin Resistance and Vitamin D Deficiency: A Link Beyond the Appearances,” *Frontiers in Cardiovascular Medicine* 9 (2022): 859793, <https://doi.org/10.3389/fcvm.2022.859793>.
- 50 Lily Nichols, *Real Food for Pregnancy: The Science and Wisdom of Optimal Prenatal Nutrition* (United States?: Lily Nichols, 2018), xx.

Chapter 16

- [1](#) Loren Cordain, *The Paleo Diet*, rev. ed. (Hoboken, NJ: John Wiley & Sons, 2011), 10.
- [2](#) Ehsan Ghaedi et al., “Effects of a Paleolithic Diet on Cardiovascular Disease Risk Factors: A Systematic Review and Meta-Analysis of Randomized Controlled Trials,” *Advances in Nutrition* 10, no. 4 (2019): 634–46, <https://doi.org/10.1093/advances/nmz007>.
- [3](#) Caroline J. Tuck, Jessica R. Biesiekierski, Peter Schmid-Grendelmeier, and Daniel Pohl, “Food Intolerances,” *Nutrients* 11, no. 7 (2019): 1684, <https://doi.org/10.3390/nu11071684>.
- [4](#) Shahir Masri, Claudia S. Miller, Raymond F. Palmer, and Nicholas Ashford, “Toxicant-Induced Loss of Tolerance for Chemicals, Foods, and Drugs: Assessing Patterns of Exposure Behind a Global Phenomenon,” *Environmental Sciences Europe* 33, (2021): 65, <https://doi.org/10.1186/s12302-021-00504-z>.
- [5](#) Qiuyu Zhang et al., “Antibiotic-Induced Gut Microbiota Dysbiosis Damages the Intestinal Barrier, Increasing Food Allergy in Adult Mice,” *Nutrients* 13, no. 10 (2021): 3315, <https://doi.org/10.3390/nu13103315>.
- [6](#) Akihito Harusato et al., “Dietary Emulsifiers Exacerbate Food Allergy and Colonic Type 2 Immune Response through Microbiota Modulation,” *Nutrients* 14, no. 23 (2022): 4983, <https://doi.org/10.3390/nu14234983>; Sabrine Naimi, Emilie Viennois, Andrew T. Gewirtz, and Benoit Chassaing, “Direct Impact of Commonly Used Dietary Emulsifiers on Human Gut Microbiota. *Microbiome* 9, no. 1 (2021): 66, <https://doi.org/10.1186/s40168-020-00996-6>.
- [7](#) Simon Kapaj, Hans Peterson, Karsten Liber, and Prosun Bhattacharya, “Human Health Effects from Chronic Arsenic Poisoning: A Review,” *Journal of Environmental Science and Health. Part A, Toxic/Hazardous Substances & Environmental Engineering* 41, no. 10 (2006): 2399–428, <https://doi.org/10.1080/10934520600873571>; Yann Malaisé et al., “Oral Exposure to Bisphenols Induced Food Intolerance and Colitis in Vivo by Modulating Immune Response in Adult Mice,” *Food and Chemical* 146 (2020): 111773, <https://doi.org/10.1016/j.fct.2020.111773>.
- [8](#) Alessio Fasano, “All Disease Begins in the (Leaky) Gut: Role of

- Zonulin-Mediated Gut Permeability in the Pathogenesis of Some Chronic Inflammatory Diseases," *F1000Research* 9 (2020): F1000 Faculty Rev-69, <https://doi.org/10.12688/f1000research.20510.1>.
- 9 Stephen J. Genuis, "Sensitivity-Related Illness: The Escalating Pandemic of Allergy, Food Intolerance and Chemical Sensitivity," *The Science of the Total Environment* 408, no. 24 (2010): 6047–61, <https://doi.org/10.1016/j.scitotenv.2010.08.047>.
- 10 Erin Smith, Amy Foxx-Orenstein, Lisa A. Marks, and Neera Agrwal, "Food Sensitivity Testing and Elimination Diets in the Management of Irritable Bowel Syndrome," *Journal of the American Osteopathic Association* 120, no. 1 (2020): 19–23, <https://doi.org/10.7556/jaoa.2020.008>.

Chapter 17

- 1 Jen Unwin and David Unwin, “A Simple Model to Find Patient Hope for Positive Lifestyle Changes: GRIN,” *Journal of Holistic Healthcare* 16 no. 2 (2019): 18–22, <https://bhma.org/wp-content/uploads/2019/06/GRIN-Unwins-JHH-16.2.pdf>; GRIN process summarized with permission.

Chapter 18

- [1](#) Miriam Kalamian, *Keto for Cancer: Ketogenic Metabolic Therapy As a Targeted Nutritional Strategy* (White River Junction Vermont: Chelsea Green Publishing, 2017).
- [2](#) Stephen Phinney and Jeff Volek, “The Ten Defining Characteristics of a Well-Formulated Ketogenic Diet,” Virta, August 13, 2018, <https://www.virtahealth.com/blog/well-formulated-ketogenic-diet>.
- [3](#) Jakob Norgren et al., “Ketosis After Intake of Coconut Oil and Caprylic Acid—With and Without Glucose: A Cross-Over Study in Healthy Older Adults,” *Frontiers in Nutrition* 7 (2020): 40, <https://doi.org/10.3389/fnut.2020.00040>.

Chapter 19

- [1](#) Gary Taubes, *Good Calories, Bad Calories* (New York: Alfred A. Knopf, 2007), 320.
- [2](#) Zsófia Clemens, “Paleolithic Ketogenic Diet (PKD) in Chronic Diseases: Clinical and Research Data,” *Journal of Evolution and Health* 3, no. 2 (2018), <http://dx.doi.org/10.15310/2334-3591.1115>.
- [3](#) Amber O’Hearn, “Can a Carnivore Diet Provide All Essential Nutrients?,” *Current Opinion in Endocrinology, Diabetes, and Obesity* 27, no. 5 (2020): 312–6, <https://doi.org/10.1097/MED.0000000000000576>.
- [4](#) Philip Mathew and Jennifer L. Pfleghaar, “Egg Allergy,” in *StatPearls* (internet) (Treasure Island, FL: StatPearls Publishing), updated July 23, 2022, <https://www.ncbi.nlm.nih.gov/books/NBK538192>.
- [5](#) “Food Allergies,” US Food and Drug Administration, last modified January 10, 2023, <https://www.fda.gov/food/food-labeling-nutrition/food-allergies>.
- [6](#) Laura Maintz and Natalija Novak, “Histamine and Histamine Intolerance,” *The American Journal of Clinical Nutrition* 85, no. 5 (2007): 1185–96, <https://doi.org/10.1093/ajcn/85.5.1185>; Roland Seifert et al., “Molecular and Cellular Analysis of Human Histamine Receptor Subtypes,” *Trends in Pharmacological Sciences* 34, no. 1 (2013): 33–58, <https://doi.org/10.1016/j.tips.2012.11.001>.

Chapter 20

- [1](#) Mamdouh Abdulrhman et al., “Effects of Honey, Sucrose and Glucose on Blood Glucose and C-Peptide in Patients with Type 1 Diabetes Mellitus,” *Complementary Therapies in Clinical Practice* 19, no. 1 (2013): 15–9, <https://doi.org/10.1016/j.ctcp.2012.08.002>.
- [2](#) Emami, Mohammad Reza et al., “Acute Effects of Caffeine Ingestion on Glycemic Indices: A Systematic Review and Meta-Analysis of Clinical Trials,” *Complementary Therapies in Medicine* 44 (2019): 282–90, <https://doi.org/10.1016/j.ctim.2019.05.003>.
- [3](#) Fawaz Alasmari, “Caffeine Induces Neurobehavioral Effects through Modulating Neurotransmitters,” *Saudi Pharmaceutical Journal* 28, no. 4 (2020): 445–51, <https://doi.org/10.1016/j.jsps.2020.02.005>.
- [4](#) Andrew W. McHill, Benjamin J. Smith, and Kenneth P. Wright, Jr., “Effects of Caffeine on Skin and Core Temperatures, Alertness, and Recovery Sleep during Circadian Misalignment,” *Journal of Biological Rhythms* 29, no. 2 (2014): 131–43, <https://doi.org/10.1177/0748730414523078>.
- [5](#) Vasilios G. Masdrakis, Manolis Markianos, and Panagiotis Oulis, “Lack of Specific Association between Panicogenic Properties of Caffeine and HPA-Axis Activation. A Placebo-Controlled Study of Caffeine Challenge in Patients with Panic Disorder.” *Psychiatry Research* 229, no. 1–2 (2015): 75–81, <https://doi.org/10.1016/j.psychres.2015.07.069>.

Appendix A

- [1](#) Esa T. Soppi, “Iron Deficiency Without Anemia: A Clinical Challenge,” *Clinical Case Reports* 6, no. 6 (2018): 1082–6, <https://doi.org/10.1002/ccr3.1529>.
- [2](#) Shrey Kumar Srivastav et al., “Serum Ferritin in Metabolic Syndrome: Mechanisms and Clinical Applications,” *Pathophysiology* 29, no. 2 (2022): 319–25, <https://doi.org/10.3390/pathophysiology29020023>.
- [3](#) Alison U. Kelly, Stephen T. McSorley, Prinesh Patel, and Dinesh Talwar, “Interpreting Iron Studies,” *BMJ Clinical Research* 357 (2017): j2513,

<https://doi.org/10.1136/bmj.j2513>.

- 4 Stella Iacovides et al., “Could the Ketogenic Diet Induce a Shift in Thyroid Function and Support a Metabolic Advantage in Healthy Participants? A Pilot Randomized-Controlled-Crossover Trial,” *PloS ONE* 17, no. 6 (2022): e0269440, <https://doi.org/10.1371/journal.pone.0269440>.
- 5 Philip N. Patsalos, Edgar P. Spencer, and Dave J. Berry, “Therapeutic Drug Monitoring of Antiepileptic Drugs in Epilepsy: A 2018 Update,” *Therapeutic Drug Monitoring* 40, no. 5 (2018): 526–48, <https://doi.org/10.1097/FTD.0000000000000546>; Shery Jacob and Anroop B. Nair, “An Updated Overview on Therapeutic Drug Monitoring of Recent Antiepileptic Drugs,” *Drugs in R&D* 16, no. 4 (2016): 303–16, <https://doi.org/10.1007/s40268-016-0148-6>.

Appendix C

- 1 Mary Ann Asson-Batres, “How Dietary Deficiency Studies Have Illuminated the Many Roles of Vitamin A During Development and Postnatal Life,” *Sub-Cellular Biochemistry* 95 (2020): 1–26, https://doi.org/10.1007/978-3-030-42282-0_1.
- 2 Marta U. Wołoszynowska-Fraser, Azita Kouchmeshky, and Peter McCaffery, “Vitamin A and Retinoic Acid in Cognition and Cognitive Disease,” *Annual Review of Nutrition* 40 (2020): 247–72, <https://doi.org/10.1146/annurev-nutr-122319-034227>.
- 3 David O. Kennedy, “B Vitamins and the Brain: Mechanisms, Dose and Efficacy—A Review,” *Nutrients* 8, no. 2 (2016): 68, <https://doi.org/10.3390/nu8020068>.
- 4 Shibani Dhir, Maya Tarasenko, Eleonora Napoli, and Cecilia Giulivi, “Neurological, Psychiatric, and Biochemical Aspects of Thiamine Deficiency in Children and Adults,” *Frontiers in Psychiatry* 10 (2019): 207, <https://doi.org/10.3389/fpsyg.2019.00207>.
- 5 Kiran Thakur et al., “Riboflavin and Health: A Review of Recent Human Research,” *Critical Reviews in Food Science and Nutrition* 57, no. 17 (2017): 3650–60, <https://doi.org/10.1080/10408398.2016.1145104>.

- 6 Thakur et al., “Riboflavin and Health.
- 7 Kennedy, “B Vitamins and the Brain,”
- 8 Janos Zempleni, Subhashinee S.K. Wijeratne, and Yousef I. Hassan, “Biotin,” *Biofactors* 35, no. 1 (2009): 36–46, <https://doi.org/10.1002/biof.8>.
- 9 Carlos Alberto Calderón-Ospina and Mauricio Orlando Nava-Mesa, “B Vitamins in the Nervous System: Current Knowledge of the Biochemical Modes of Action and Synergies of Thiamine, Pyridoxine, and Cobalamin,” *CNS Neuroscience & Therapeutics* 26, no. 1 (2020): 5–13, <https://doi.org/10.1111/cns.13207>.
- 10 Peter Lyon, Victoria Strippoli, Byron Fang, and Luisa Cimmino, “B Vitamins and One-Carbon Metabolism: Implications in Human Health and Disease,” *Nutrients* 12, no. 9 (2020): 2867, <https://doi.org/10.3390/nu12092867>.
- 11 Karin Amrein et al., “Vitamin D Deficiency 2.0: An Update on the Current Status Worldwide,” *European Journal of Clinical Nutrition* 74, no. 11 (2020): 1498–513, <https://doi.org/10.1038/s41430-020-0558-y>.
- 12 Tyler C. Huff et al., “Vitamin C Regulates Schwann Cell Myelination by Promoting DNA Demethylation of Pro-Myelinating Genes,” *Journal of Neurochemistry* 157, no. 6 (2021): 1759–73, <https://doi.org/10.1111/jnc.15015>.
- 13 Phoebe E. Mayne and Thomas H. J. Burne, “Vitamin D in Synaptic Plasticity, Cognitive Function, and Neuropsychiatric Illness,” *Trends in Neurosciences* 42, no. 4 (2019): 293–306, <https://doi.org/10.1016/j.tins.2019.01.003>.
- 14 Mónica López-Vicente et al., “Maternal Circulating Vitamin D₃ Levels During Pregnancy and Behaviour across Childhood,” *Scientific Reports* 9, no. 1 (2019): 14792, <https://doi.org/10.1038/s41598-019-51325-3>.
- 15 Jean-Marc Zingg, “Vitamin E: Regulatory Role on Signal Transduction,” *IUBMB Life* 71, no. 4 (2019): 456–78, <https://doi.org/10.1002/iub.1986>.
- 16 Daniela-Saveta Popa, Galya Bigman, and Marius Emil Rusu, “The Role of Vitamin K in Humans: Implication in Aging and Age-Associated Diseases,” *Antioxidants* 10, no. 4 (2021): 566, <https://doi.org/10.3390/antiox10040566>.
- 17 Ludovico Aliso et al., “The Relationships Between Vitamin K and

- Cognition: A Review of Current Evidence," *Frontiers in Neurology* 10 (2019): 239, <https://doi.org/10.3389/fneur.2019.00239>.
- 18 Katarzyna Maresz, "Growing Evidence of a Proven Mechanism Shows Vitamin K2 Can Impact Health Conditions Beyond Bone and Cardiovascular," *Integrative Medicine* 20, no. 4 (2021): 34–8.
- 19 Paweł Mozolewski et al., "The Role of Nuclear Ca²⁺ in Maintaining Neuronal Homeostasis and Brain Health," *Journal of Cell Science* 134, no. 8 (2021): jcs254904, <https://doi.org/10.1242/jcs.254904>.
- 20 Pramod Sukumaran et al., "Calcium Signaling Regulates Autophagy and Apoptosis," *Cells* 10, no. 8 (2021): 2125, <https://doi.org/10.3390/cells10082125>.
- 21 Xabier Elorza-Vidal, Héctor Gaitán-Peña, and Raúl Estévez, "Chloride Channels in Astrocytes: Structure, Roles in Brain Homeostasis and Implications in Disease," *International Journal of Molecular Sciences* 20, no. 5 (2019): 1034, <https://doi.org/10.3390/ijms20051034>.
- 22 Steven H. Zeisel and Kerry-Ann da Costa, "Choline: An Essential Nutrient for Public Health," *Nutrition Reviews* 67, no. 11 (2009): 615–23, <https://doi.org/10.1111/j.1753-4887.2009.00246.x>; Jan Krzysztof Blusztajn, Barbara E. Slack, Tiffany J. Mellott, "Neuroprotective Actions of Dietary Choline," *Nutrients* 9, no. 8 (2017): 815, <https://doi.org/10.3390/nu9080815>.
- 23 Cheri M. Ackerman and Christopher J. Chang, "Copper Signaling in the Brain and Beyond," *The Journal of Biological Chemistry* 293, no. 13 (2018): 4628–35, <https://doi.org/10.1074/jbc.R117.000176>.
- 24 Marilu Jurado-Flores, Firas Warda, and Arshag Mooradian, "Pathophysiology and Clinical Features of Neuropsychiatric Manifestations of Thyroid Disease," *Journal of the Endocrine Society* 6, no. 2 (2022): bvab194, <https://doi.org/10.1210/jendso/bvab194>.
- 25 Adrienne Hatch-McChesney and Harris R. Lieberman, "Iodine and Iodine Deficiency: A Comprehensive Review of a Re-Emerging Issue," *Nutrients* 14, no. 17 (2022): 3474, <https://doi.org/10.3390/nu14173474>.
- 26 Nadia Sawicka-Gutaj, Natalia Zawalna, Paweł Gut, and Marek Ruchała, "Relationship between Thyroid Hormones and Central Nervous System Metabolism in Physiological and Pathological Conditions," *Pharmacological Reports* 74, no. 5 (2022): 847–58, <https://doi.org/10.1007/s43440-022-00377-w>.

- 27 Ana Ferreira, Pedro Neves, and Raffaella Gozzelino, “Multilevel Impacts of Iron in the Brain: The Cross Talk between Neurophysiological Mechanisms, Cognition, and Social Behavior,” *Pharmaceuticals* 12, no. 3 (2019): 126, <https://doi.org/10.3390/ph12030126>.
- 28 Michael K. Georgieff, “Iron Deficiency in Pregnancy,” *American Journal of Obstetrics and Gynecology* 223, no. 4 (2020): 516–24, <https://doi.org/10.1016/j.ajog.2020.03.006>.
- 29 Ryu Yamanaka, Yutaka Shindo, and Kotaro Oka, “Magnesium Is a Key Player in Neuronal Maturation and Neuropathology,” *International Journal of Molecular Sciences* 20, no. 14 (2019): 3439, <https://doi.org/10.3390/ijms20143439>.
- 30 Rekha C. Balachandran et al., “Brain Manganese and the Balance between Essential Roles and Neurotoxicity,” *The Journal of Biological Chemistry* 295, no. 19 (2020): 6312–29, <https://doi.org/10.1074/jbc.REV119.009453>; Mani Ratnesh S. Sandhu et al., “Astroglial Glutamine Synthetase and the Pathogenesis of Mesial Temporal Lobe Epilepsy,” *Frontiers in Neurology* 12 (2021): 665334, <https://doi.org/10.3389/fneur.2021.665334>.
- 31 Steven M. Chrysafides, Stephen Bordes, and Sandeep Sharma, “Physiology, Resting Potential,” in *StatPearls* (Treasure Island, FL: StatPearls Publishing, 2022 Jan-), updated April 21, 2021, <https://www.ncbi.nlm.nih.gov/books/NBK538338>.
- 32 Daniel J. Torres, Naghum Alfulaij, and Marla J. Berry, “Stress and the Brain: An Emerging Role for Selenium,” *Frontiers in Neuroscience* 15 (2021): 666601, <https://doi.org/10.3389/fnins.2021.666601>.
- 33 Torres, Alfulaij, and Berry, “Stress and the Brain.”
- 34 Alberto Granzotto, Lorella M. T. Canzoniero, and Stefano L. Sensi, “A Neurotoxic *Ménage-à-trois*: Glutamate, Calcium, and Zinc in the Excitotoxic Cascade,” *Frontiers in Molecular Neuroscience* 13 (2020): 600089, <https://doi.org/10.3389/fnmol.2020.600089>; Rebecca F. Krall, Thanos Tzounopoulos, and Elias Aizenman, “The Function and Regulation of Zinc in the Brain,” *Neuroscience* 457 (2021): 235–58, <https://doi.org/10.1016/j.neuroscience.2021.01.010>.